A HANDBOOK OF CLINICAL MEDICINE

VOLUME ONE

DISEASES OF THE
HEART AND KIDNEY

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U. N. DHUR & SONS, LIMITED.

15, BANKIM CHATTERJEE ST., CALCUTTA.

1947

First Edition *1947*

Published by D. N. Dhur, B.L., of Messrs. U. N. Dhur & Sons Ltd., 15, Bankim Chatterjee St., Calcutta, and printed by J. N. Bose at the Indian Press Ltd., 93-A, Dharamtala St., Calcutta.

PREFACE

Despite the existence of many excellent text-books of clinical medicine, we have felt the necessity of bringing out a publication on this important subject in small separate volumes, dealing with its different branches in a manner that has often been found to be helpful at the bedside, both in the teaching of medical students and in the taking up and management of patients by the general practitioners.

Our plea for giving the priority of publication to this present volume on cardiovascular and renal diseases is firstly because this branch of medicine is often considered both by the students and the practitioners to be too complex to be easily grasped and secondly because cardio-renal disturbances are exceedingly common in clinical practice, demanding their urgent attention.

The volume opens with an introduction on definition of disease and certain other terms, signs and symptoms, mechanism of their production, a general scheme of case-taking for routine use in all cases and a special scheme for cardiac cases.

It has incorporated in detail the various essential clinical methods of examination of the heart and the kidneys. Ætiology, pahology, symptomatology, diagnosis and treatment have been briefly considered to give the reader a clear picture of the disease processes and their management. Unnecessary and controversial details have been deliberately eschewed, as far as practicable.

Throughout the book, stress has been laid on considering the diseases of the heart from the ætiological and functional stand point

and not from the anatomical view point only. Hence vascular diseases have not been allotted a separate chapter.

The volume ends with two appendices dealing with the basic essentials of clinical electrocardiography and the methods of urine-analysis respectively.

Finally, we hope this small book would prove useful to those for whom it is intended.

J. C. B.
P. K. C.

Calcutta
Dated, the 7th December 1946.

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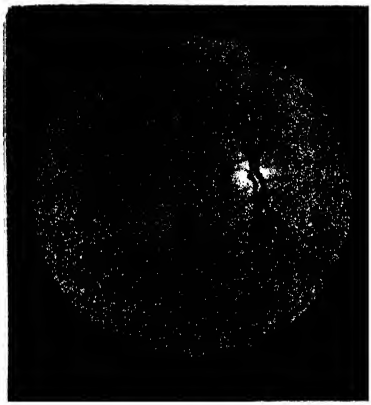

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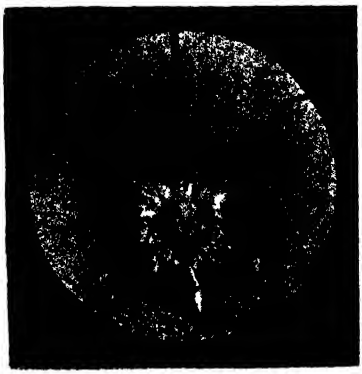

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A HANDBOOK OF CLINICAL MEDICINE

PART I

GENERAL INTRODUCTION

Disease, its definition.—Disease is a disturbance of the sense of well-being, both physical and mental. Such a state of well-being is maintained in health by the anatomical and functional integrity of the various organs and tissues, as well as by the capacity of the mind and body, to adapt themselves to the varying environments to which the individual is exposed in his day-to-day struggle for existence.

Aetiology.—The causes which produce diseases are called ætiological factors. These are :

1. Infections by (*a*) Bacteria, (*b*) Parasites, and (*c*) Filterable viruses.
2. Intoxication by toxins of (*a*) Bacterial origin, (*b*) Metabolic origin, or (*c*) Chemical poisons.
3. Deficiency in vitamins.
4. Metabolic derangement.
5. Developmental errors and hereditary diatheses.
6. Endocrine dysfunction.
7. Neoplasm.
8. Injury from (*a*) Mechanical causes, *e.g.*, trauma, (*b*) Physical causes—heat, electricity, (*c*) Chemicals—corrosive acids and alkalies.
9. Psychological causes.

Pathology.—The word pathology literally means science of disease. It is, however, used in a more restricted sense to mean the study of changes in the organs and tissues brought about by one or more of the ætiological factors. Such changes may be local or general.

The local changes may be

1. Inflammation and its sequelæ, such as, (*a*) Suppuration, (*b*) Degeneration, (*c*) Necrosis, (*d*) Caseation, (*e*) Granulation, (*f*) Cicatrisation.
2. Degeneration and necrosis from other causes, and gangrene.

3. Circulatory disturbances, e.g., (*a*) Congestion—active or passive, (*b*) Thrombosis, (*c*) Infarction.

4. Neoplasm.

The general changes are chiefly of two types—

1. Metabolic disturbances resulting in fever, wasting, acidosis etc.

2. Alteration in tissue response in general, to foreign irritants, *e.g.*, allergy, hypersensitiveness, immunity and tolerance.

Symptoms and signs.—*Symptoms* are subjective sensations produced by a disturbance of functions of one or more organs due to one or more ætiological factors. The disturbance of functions may be (*a*) over-action, (*b*) under-action, (*c*) altered action, and (*d*) loss of function.

The subjective sensations may arise as a result of abnormal excitation of nerves by the local changes in the tissues. These impulses may cause abnormal sensations of discomfort directly where sensory nerves exist or they may cause referred sensations or reflex disturbances. For instance, an inflammation in the middle ear causes earache, whereas an inflammation in the gall-bladder causes aching in the tip of the right shoulder as referred pain.

Symptoms may also arise as a result of abnormal reaction of organs to psychogenic disturbances.

Signs are objective evidences of disease produced by altered anatomy and physiology of organs and tissues. They are capable of demonstration. Signs may be produced in several ways as follows:

1. Alteration in size and shape of organs *e.g.*, enlargement of the heart in heart failure as detected by palpation of apex-beat and percussion of the borders; irregularities in an enlarged liver as in malignant disease.

2. Disturbances of nervous functions either motor, sensory or psychological, and alteration in reflexes, such as in diseases of the nervous system.

3. Alteration of secretions and excretions of the body, such as excessive expectoration or urinary abnormalities.

4. Alteration in the chemistry of blood and tissue fluids. Such changes lead to, or indicate, a disturbance of metabolism and/or of the heat-regulating mechanism. The so-called constitutional signs like wasting, fever, malaise, dehydration, anæmia etc., or signs of a more specific character such as tetany of hypocalcæmia, may arise from such causes.

Diagnosis.—The first duty of a physician is to relieve or prevent the suffering of his patient. This object can however be attained by arriving at a complete and accurate diagnosis as to the nature, site and extent of the anatomical and physiological disturbances caused by the disease as well as its ætiological factors. A complete diagnosis therefore, must be anatomical, functional and ætiological.

For this purpose, the physician collects all the data regarding the evolution of the symptoms and their character, the susceptibilities of the patient and evidences of changes in the organs. The clinical approach for the elicitation of all these data and detection of physical signs constitute case-taking. Sometimes to confirm or supplement the physical signs, laboratory or instrumental aid is necessary, such as the chemical analysis of urine, blood, cerebrospinal fluid etc., microscopical and bacteriological examinations, use of the X-rays, electro-cardiography, endoscopy etc. A diagnosis may not always be arrived at even after all these investigations and recourse has to be taken to biopsy (pathological and histological examination of tissues removed from the body during life) or exploratory operations. Inspite of the adoption of the above methods, the diagnosis may still remain obscure, and in the last resort, may be arrived at or confirmed by an autopsy. Although this does not help in the performance of the first duty of the physician as a healer of suffering, it adds considerably to our knowledge of diseases and helps us in improving our diagnostic acumen in future cases.

Case-taking.—In case-taking a definite scheme of investigations is followed to ensure thoroughness. It consists of three parts, as follows:

1. History taking and interrogation of the patient.
2. Physical examination (*a*) general and (*b*) systemic.
3. Special investigations with laboratory or instrumental aid.

General Scheme of Case-taking

History taking

1. **Name, age, sex, race, caste, religion, occupation, address and date of examination.**

2. **Complaints.**—These should be recorded, as far as possible, in the patient's own words ; *i.e.,* the description, in short, of the character of the chief subjective sensations of the patient. The duration of each complaint should be noted.

3. **History of the present illness.**—By careful interrogation or from the patient's description, the details regarding symptoms are elicited, such as mode of onset, chronological sequence of their development, continuous or in paroxysms, causes of aggravation or relief etc. In these enquiries, it is necessary to avoid leading questions.

4. **History of past illnesses.**—Enquiries should be made for (*a*) Past diseases of which the present symptoms may be after-effects, (*b*) Diseases which may predispose to the suspected present condition, (*c*) Past illnesses which may be early manifestations of the present disease, such as exudative pleurisy preceding pulmonary tuberculosis, (*d*) Any other disease of importance in the past life.

5. **Family history.**—(*a*) A general enquiry as to the condition of health in the nearest relations, if alive, or the mode of their death, if dead.

(*b*) In case of infectious diseases, an enquiry as to similar infection in other members of the family.

(*c*) An enquiry as to the presence of similar symptoms and signs in other members, e.g., in diabetes mellitus, epidemic dropsy, familial acholuric jaundice, hæmophilia, epilepsy, hereditary ataxias etc.

6. **Personal history.**—Enquiries should be made into (*a*) Environmental conditions, whether hygienic or unhygienic.

(*b*) Habits, sedentary or active.

(*c*) Mental make-up—whether habitually jovial, depressive, anxious or restless etc.

(*d*) Nature of food and hour of meals.

(*e*) Use of tobacco, tea, coffee or alcohol, specially the quantities taken in twentyfour hours.

(*f*) Nature of work involved in the occupation.

(*a*) Marital state, number of children; in case of females, miscarriages or abortions, menstrual history.

PHYSICAL EXAMINATION

I. **General examination.**—The following should be noted:—

(*a*) State of consciousness and intelligence, (*b*) Physical build —development in proportion to age, (*c*) State of nutrition—weight of the patient.

(*d*) General attitude.—Restless, apprehensive, distressed or dull and apathetic. If the patient is in bed, note the decubitus or the position of patient in bed, e.g., dorsal, lateral, propped up or stooping forward.

(*e*) Facies or facial expression. (*f*) Pallor, jaundice, cyanosis, anasarca, rash or spots on the body.

(*g*) Any marked abnormality in eyes, e.g., exophthalmos; in the neck e.g., engorged veins, pulsating carotids or goitre etc.

(*h*) Enlarged neck glands.

(*i*) Any marked change in the extremities like clubbing of fingers and toes, œdema of feet.

(*j*) Temperature. (*k*) Pulse. (*l*) Respiration.

II. **Systemic examination.**—The system to be examined first and most elaborately will be suggested by the presenting symptoms. The other systems are to be examinqd more briefly according to the routine procedure and any abnormal finding is to be recorded.

1. **Circulatory system.**—(*a*) *Subjective symptoms.*—Dyspnœa on exertion, præcordial pain, palpitation.

(*b*) *Objective signs.* (i) *Inspection.*—Shape of præcordium, site of apical impulse, other pulsations in the præcordium and outside.

(ii) *Palpation.*—Site and character of the apical impulse, other pulsations, thrills, if any.

(iii) *Percussion.*—Site of the apex, the left, right and upper borders of heart, cardio-hepatic angle.

(iv) *Auscultation.*—Study of the cardiac sounds—character, intensity, duration and distinctness. Murmurs, if present, note site of maximum intensity, timing, character, propagation, pericardial friction rub. Auscultate successively in the mitral, tricuspid, pulmonary and aortic areas and record findings in each area.

Vessels.—Radial pulse—character, rate, volume, tension and condition of the arterial wall. Look for abnormal prominence, tortuosity, irregularities and hardness of the brachial, temporal and dorsalis pedis arteries.

Blood pressure.—Systolic and diastolic.

2. **Respiratory system.**—(*a*) *Subjective symptoms.*—Chest pain, cough, expectoration, hæmoptysis, dyspnœa.

(*b*) *Objective signs.*—(i) *Inspection.*—Upper respiratory passages—tonsils, nasal sinuses. Shape of the chest, wasting of muscles, respiratory movements, rate of respiration.

(ii) *Palpation.*—Shape and movements of chest, vocal fremitus, local tenderness. Irritability of the muscles—myotatic irritability.

(iii) *Percussion.*—Extent of the borders diminished or increased. Abnormalities of resonance over the lungs and sense of resistance to the percussing finger.

(iv) *Auscultation.*—Breath sounds and their abnormalities, adventitious sounds if any, vocal resonance.

3. **Alimentary system.**—(*a*) *Subjective symptoms.*—Anorexia, flatulence, pain or colic, acid eructations, heartburn, nausea, vomiting, hæmatemesis, melæna, diarrhœa, constipation.

(*b*) *Objective signs.*—(i) *Inspection.*—Tongue, teeth and gums. Abdomen—movements with respiration, abnormal distension or retraction, prominent veins with direction of blood flow. Localised swellings, visible peristalsis—site of origin and mode of spread.

(ii) *Palpation.*—Abdomen.—Movement with respiration, soft or rigid—local or general. Tenderness, localised or generalised. Abdominal masses if any. Palpation of liver, spleen, ascending and iliac colons.

(iii) *Percussion.*—Abdomen.—Localised dullness, shifting dullness, fluid thrills. Percussion of different organs.

(iv) *Auscultation* for peristaltic sounds.

(v) *Rectal examination.*

4. **Urinary system.** (*a*) *Subjective symptoms.*—Frequency of micturition—diurnal or nocturnal, oliguria, hæmaturia, dysuria.

(*b*) *Objective signs.*—(i) *Palpation.*—Palpation of the kidneys, enlargement if any and tenderness in loins.

Urine examination.

5. **Nervous system.**—(*a*) *Subjective symptoms.*—Paralysis, paresis, ataxia, tremor, anæsthesia, paræsthesia, headache, disturbance of consciousness, memory, judgment, sleep etc.

(*b*) *Objective signs.*—Psychological functions, speech, gait, cranial nerve functions, motor functions, sensory functions, reflexes (superficial and deep), visceral reflexes, trophic changes.

(*c*) *Ophthalmoscopic examination* of the fundus of the eye.

PART II

CARDIOVASCULAR SYSTEM

CHAPTER I

INTRODUCTION, INTERROGATION AND CASE-TAKING

General considerations.—The heart consists of three parts, the myocardium, the endocardium and the pericardium. The myocardium supplies the driving force for the circulation and certain parts in it, such as the sinoauricular and the auriculoventricular nodes and the bundle of His, are concerned with the origin and spread of the cardiac impulse, thus keeping up the normal rhythm. The endocardium is thrown into folds at the cardiac orifices and forms valves to maintain the circulation in one direction. The pericardium encloses between its layers a lubricated space providing the heart a smooth and frictionless contact with the surroundings.

It is obvious, therefore, that the myocardium plays the most important part in the main function of the heart; and myocardial diseases affect cardiac efficiency directly, leading to symptoms by reduction of cardiac reserve.

Valvular diseases may cause a leakage of blood in abnormal direction or an abnormal narrowing of the orifices, thus increasing the mechanical burden on the myocardium. Such defects, however, may be suffciently compensated for, and may in no way reduce the cardiac efficiency provided the myocardium is healthy. Their significance lies in so far as they increase the burden on the myocardium already damaged.

Pericardial diseases obtain importance when they produce a mechanical strain on the heart either by anchoring it with the rigid chest wall, or by compressing it with effusion or contracting cicatrix.

Causes of heart disease.—1. Rheumatic infection. 2. Hypertension, essential or nephritic. 3. Coronary arteriosclerosis. 4. Syphilis. 5. Chronic pulmonary disease causing increased resistance and anoxæmia (Cor pulmonale). 6. Thyrotoxicosis. 7. Psychoneurosis, Neurocirculatory asthenia. 8. Toxic, metabolic, nutritional and deficiency factors as in epidemic dropsy, beriberi. 9. Congenital defects.

Investigation and Case-taking.—Patient's name, race, religion and address are noted.

Age.—Common causes of heart disease at different ages are

(*a*) Infancy—Congenital defect.

(*b*) Childhood and adolescence (upto 20 years)—Rheumatic infection.

(*c*) Adults (20 to 35 years)—Chronic rheumatic valvular disease, neurocirculatory asthenia, thyrotoxicosis.

(*d*) Middle age (40 to 50 years)—Hypertension, coronary sclerosis, emphysema and chronic bronchitis, syphilis.

(*e*) Old age (above 55 years)—Coronary sclerosis, hypertension (rare).

Sex.—Syphilitic heart disease and coronary sclerosis are distinctly rare in females. Thyrotoxicosis is more common in females. Contrary to the common belief, rheumatic heart disease is more commonly seen in males than in females. Pregnancy and labour provide additional problems in females with heart disease.

Occupation.—The nature of occupation is important in assessment of prognosis.

Complaints. 1. *Dyspnoea.*—This is a consciousness of the effort of breathing causing discomfort. Dyspnœa may be mainly of three varieties,—exertional, continuous and paroxysmal.

(*a*) *Exertional dyspnoea.*—In the early stages. dyspnœa occurs on accustomed exertion. Later, dyspnœa may be so severe as to occur on slight movements.

(*b*) *Continuous dyspnoea.*—It occurs even at rest. In extreme cases the dyspnœa is severe enough to make the patient sit up for breath (*orthopnoea*). In sitting posture the comparative freedom of diaphragmatic movements affords some relief to respiration.

(*c*) *Paroxysmal dyspnoea or cardiac asthma.*—It occurs at night when the patient is suddenly roused with intense dyspnœa. Seen in cases of (i) hypertension, (ii) aortic valve disease, (iii) coronary sclerosis, (iv) mitral stenosis (rarely).

Dyspnœa in any form is mostly seen in left ventricular failure or in mitral stenosis. It is mainly due to stasis and congestion in the pulmonary capillaries reflexly stimulating the respiratory centre.

The cause of cardiac asthma is not properly understood. It is seen in early stages of left ventricular failure with an overacting right ventricle.

2. *Praecordial pain.*—It is mainly due to three causes, *viz.*, (*a*) effort angina, (*b*) coronary thrombosis, and (*c*) neurocirculatory asthenia. The character of pain in these conditions is shown in Table I.

Other causes of præcordial pain of less importance are (*a*) syphilitic aortitis or aneurysm of aorta, (*b*) pericarditis, (*c*) congestive cardiac failure (a sense of discomfort), (*d*) reflex pain from other organs like gall bladder or stomach.

3. *Palpitation.*—A consciousness of heart beat occurs in rapid action, forcible or overaction and irregular action of the heart. This may or may not be associated with organic heart disease. More often it is due to nervous or emotional causes or due to flatulence.

TABLE I

	Effort angina.	Coronary thrombosis.	Neurocirculatory asthenia.
1. Site.	Retrosternal.	Restrosternal, rarely in epigastrium.	Mammary or submammary region.
2. Exciting causes and relation to effort.	Exertion, excitement, heavy meals and exposure to cold. Aggravated by exertion and relieved by rest.	No exciting cause. Occurs at rest. Not affected by exertion.	More or less constant pain. No definite relation to effort.
3. Radiation.	To the arm and forearm on the left, rarely to right side.	Same as in effort angina.	From inframammary to the left subscapular region.
4. Character.	Sense of constriction, oppression or choking sensation (Angina means feeling of strangulation).	Same as in effort angina. Continuous and relentless pain.	Sharp stabbing pain or aching.
5. Duration.	A few seconds to a few minutes. Rarely 10 minutes, maximum limit half-an-hour.	Continuous for hours or even days.	Continuous with varying intensity over longer periods.
6. Mode of relief.	Rest and inhalation of amyl nitrite.	Not relieved by amyl nitrite or rest. Relieved by morphine.	Not relieved by amyl nitrite or rest. Relieved by sedatives.

4. *Syncope.*—This may vary in degree from momentary giddiness to actual fainting with loss of consciousness due to cerebral anæmia. Two types of syncope are seen—*vascular* and *cardiac.*

(*a*) *Vascular syncope.*—It is more common and less often associated with organic heart disease. Peripheral vascular dilatation with consequent circulatory stasis and fall of blood pressure leads to diminished venous return, insufficient cardiac output and cerebral anæmia. The causes of vascular syncope are:—

(*i*) *Postural.*—In debilitated persons of low muscular tone and lax abdominal wall, sudden assumption of erect posture from a supine position, causes stasis of blood in the splanchnic capillaries. A momentary dizziness, or a loss of consciousness in exaggerated cases, occurs.

(*ii*) *Vasovagal.*—A reflex stimulation of vagal centre and inhibition of vasomotor centre cause a fall of blood pressure, slowing of pulse, nausea and sweating. The patient feels a sense of unsteadiness, blurring of vision and loses consciousness for periods of 2 to 10 minutes. This is usually seen in persons of poor muscular and vascular tone and debility, but may also occur in healthy people. Exciting causes are physical discomfort, intense pain, sudden emotional shock etc.

(*iii*) *Severe haemorrhage.*

(*b*) *Cardiac syncope.*—Cerebral anæmia occurs as a result of ventricular arrest, very slow or extremely rapid ventricular rate and ventricular fibrillation. In complete heart-block, if the ventricular rate becomes very slow (below 20 per minute) or when there is temporary cessation, loss of consciousness occurs if the interval between ventricular beats exceeds 5 to 10 seconds. Epileptiform fits may occur in longer intervals. Cases of repeated syncope with epileptiform convulsions, but without biting of tongue and involuntary evacuation of bowels and bladder, occurring in cases of heart block are called Adams-Stokes syndrome.

5. *Swelling of the legs.*—In the early stages it occurs around the ankles in the evening, disappearing with night's rest.

6. *Other symptoms* referable to

(*a*) *Nervous system.*—Headache, insomnia, failing memory, delirious state, clouding of consciousness.

(*b*) *Gastro-intestinal system.*—Anorexia, flatulence and right hypochondriac pain.

Past illness.—Enquiries should be made for the previous history of

1. Rheumatic infection and its various manifestations, such as (*a*) irregular fever, sore throat, fleeting pain in limbs and joints, swelling of big joints, (*b*) cutaneous nodules, (*c*) chorea.

2. Syphilis. (*a*) Exposure to infection, (*b*) Penile scar, (*c*) Abortion or miscarriage in wife, (*d*) Sore throat, cutaneous eruption, glandular enlargement.

3. Hypertension. (*a*) Essential, (*b*) Nephritic—oliguria, hæmaturia, œdema.

4. Chronic bronchitis, asthma, etc.

5. Epidemic dropsy.

6. Acute infective fevers like diphtheria and influenza.

7. Beriberi.

Family history.—(*a*) Rheumatic heart disease, (b) hypertensive and arteriosclerotic disease, (*c*) epidemic dropsy, (*d*) sudden death with or without anginal pain or paralysis suggesting coronary thrombosis or apoplexy should be asked for.

Personal history.—Habits of the patient should be noted. Patients with sedentary habits may suffer from dyspnœa with much less exertion than one with active habits. Severity of symptoms may thus be judged. Persons of anxious worrying type and of a nervous predisposition are susceptible to functional heart disease or liable to exaggerate symptoms of existing organic disease.

Amount of tea, tobacco, coffee or alcohol consumed should be noted. The environmental condition should also be noted. Overcrowding, dampness and malnutrition predispose children to rheumatic infection.

General examination.—The following points are to be noted.

1. *State of consciousness and psychological functions.*—In severe congestive cardiac failure, there may be clouding of consciousness, confusional delirium and lack of concentration. Memory and judgment are also defective in cerebral arteriosclerosis and cardiac failure.

II. *General development, build and nutrition.*—1. Stunted growth may be due to congenital heart disease or chronic valvular disease from early childhood.

2. Heavily built obese persons with plethoric habitus are predisposed to high blood pressure.

III. *Decubitus and general attitude.*—In congestive cardiac failure the patient is propped up due to dyspnœa. In severe cases, orthopnœa is present.

IV. *Pallor.*—In heart disease, severe pallor is due to active rheumatic infection or subacute bacterial endocarditis. Severe anæmia due to other causes may itself be the cause of cardiac symptoms.

V. *Cyanosis.*—It is a blue colouration of skin and mucous membrane due to presence of an excess of reduced hæmoglobin (5 gms. or over per 100 c.c.) in the capillaries. In severe anæmia with hæmoglobin below 30 per cent. cyanosis does not occur.

The excess of reduced hæmoglobin may occur in two ways:

1. Excessive dissociation of oxygen in the capillaries due to stasis of blood and sluggish circulation (*peripheral cyanosis*).

2. Entry of reduced hæmoglobin into the systemic circulation due to (*a*) diminished oxygenation in the lungs, or (*b*) shunting of venous blood to the arterial side without passing through the lungs (*central cyanosis*).

Peripheral cyanosis is most marked in the extremities and is associated with coldness and lividity of the parts. It may be seen in: (1) exposure to cold, (2) peripheral circulatory failure, (3) Raynaud's phenomenon, (4) venous stasis as in right sided cardiac failure.

Central cyanosis is independent of the velocity of blood flow and is not associated with any coldness of the extremities. The colour

of the skin and mucous membrane is bluish, more intense and widespread.

The causes of central cyanosis are as follows:

(1) Obstruction to air passages. (2) Reduction in the area of oxygenation in the lungs as in emphysema, lobar pneumonia, bronchopneumonia, pulmonary œdema with or without left ventricular failure. (3) Direct shunting of venous blood from the right to the left side of the heart without passing through the lung: as in various congenital defects of the heart and large vessels. (4) Alteration of hæmoglobin, such as sulphæmoglobinæmia, methæmoglobinæmia, carbon monoxide poisoning. (5) Polycythæmia.

VI. *Jaundice.*—It is not a common sign in heart disease. Its presence is, however, indicative of pulmonary infarction.

VII. *Œdema.*—In the early stages of congestive failure, pitting œdema of the dependent parts, such as around the ankles in the evening, is the characteristic feature in ambulant patients. General anasarca is rare except in severe cases of congestive failure.

VIII. *Face and eyes.*—Obvious exophthalmos is indicative of thyrotoxicosis. Inequality of the pupils may be seen in aneurysm of the aorta due to pressure on the superior cervical ganglion of the sympathetic.

Subconjunctival hæmorrhage may occur in subacute bacterial endocarditis. A malar flush is said to be characteristic of mitral stenosis.

IX. *Neck.*—1. *Prominent engorged neck veins* indicate marked increase of venous pressure due to right sided congestive cardiac failure, or obstruction to the superior vena cava due to pressure by aneurysm and mediastinal growth or due to constrictive pericarditis.

Engorgement only above the level of the manubrium should be taken as pathological. If the patient is lying on a pillow, prominent veins may be seen at the root of the neck up to a height about the same level as the manubrium. This is normal; but engorgement beyond this height is pathological. The engorged veins may pulsate high in the neck in case of congestive cardiac failure, especially with tricuspid regurgitation, but not in cases of engorgement due to obstruction in the superior mediastinum. Venous pulsation is visible, but not palpable distinguishing it from carotid pulsation which is both visible and palpable. It is well to remember that venous pulsation may be seen at the root of the neck in normal young persons in the supine position.

2. *Exaggerated carotid pulsation.*—A visible and palpable pulsation can normally be seen over the common carotid arteries specially in rapid and forcible cardiac action. In exaggerated form,

it is seen in (*a*) aortic regurgitation, (*b*) severe anæmia, (*c*) thyrotoxicosis, (*d*) high blood pressure, (*e*) aneurysmal dilatation.

3. *Size of the thyroid gland.*—Enlargement may be associated with toxic cardiac symptoms.

X. *Extremities.*—These should be examined for

1. *Clubbing of the fingers and toes.*—This is shown by a bulbous enlargement of the soft tissues at the tip of the fingers or toes, and abnormal convexity of the nails, longitudinal and transverse. This is due to proliferation of the subungual connective tissue as a result of long-standing anoxæmia and toxæmia. Clubbing is seen in the following conditions.

(*a*) Cardiac causes—(*i*) Cyanotic group of congenital heart disease. (ii) Subacute bacterial endocarditis.

(b) Respiratory diseases—(*i*) Bronchiectasis. (*ii*) Chronic lung abscess. (*iii*) Chronic empyema. (*iv*) Emphysema. (*v*) Chronic fibrocaseous tuberculosis.

(*c*) Long-standing biliary cirrhosis of liver.

(*d*) Congenital defect.

In severe cases, swelling due to subperiosteal bone formation also affects the clavicles, lower ends of radius, ulna, tibia and fibula; the condition being known as chronic hypertrophic pulmonary osteoarthropathy of Charcot.

2. *Œdema.*—This is specially seen over the ankles in early stages.

3. *Rheumatic nodules.*—These are painless, firm, subcutaneous nodules of varying size, seen in fascia, ligaments or tendons around bony prominences. These should be looked for in front of the wrist, behind the elbow, over the mastoid process, around the knees and ankles. They are seen in subacute rheumatic infection of children and are indicative of activity of the infection. (See Fig. 9, page 44).

4. *Osler's nodes.*—Painful raised red nodules in the pads of the fingers and toes are found in subacute bacterial endocarditis.

5. *Petechial haemorrhage* in the skin—commonly seen in subacute bacterial endocarditis.

Systemic examination.—The examination of the cardiovascular system consists of examination of the heart, radial pulse, blood pressure and exercise tolerance.

Heart—The surface relations of the heart are shown in the figure. (Fig. No. 1). The part of the anterior chest wall overlying the heart is called the præcordium. Its boundaries are as follows:—

On the right, a curved line joining the right third and the seventh chondrosternal junctions, reaching a maximum distance of 1½" from the middle line.

On the left, a line joining a point in the left second intercostal space just internal to the left parasternal line, with the apex in the left fifth intercostal space just internal to the midclavicular line.

The *upper and lower borders* can be obtained by joining the upper and lower limits of the right and left borders.

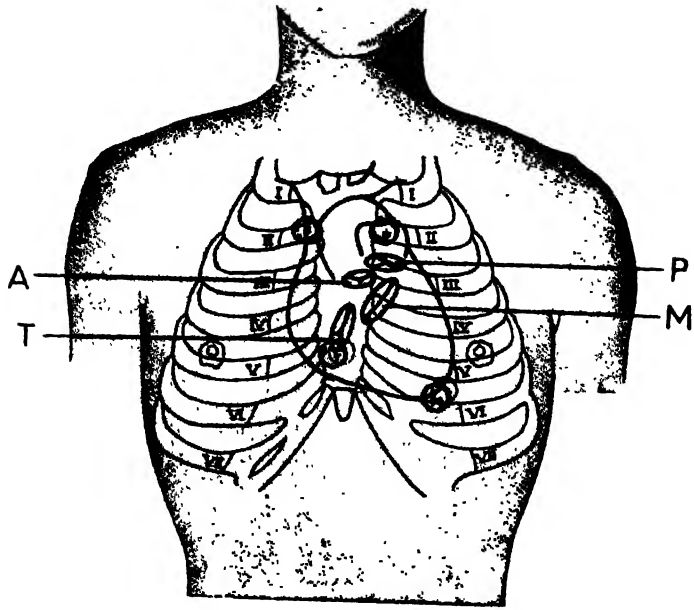


Fig. 1. The surface relations of heart and the cardiac valves. The red circles indicate the position of auscultatory areas. P—Pulmonary. M—Mitral. T—Tricuspid. A—Aortic.

The surface markings of the different cardiac valves are shown in the figure. In auscultation for the sounds produced at the different valves, the stethoscope is placed over areas of the chest wall where the chambers in which these valves lie are most superficial. These areas are named after the corresponding valves; and sounds produced in that particular valve or orifice, are heard best in that area. These areas do not correspond with the anatomical position of the valves. These auscultatory areas are (1) *mitral area,* over the cardiac apex, (2) *tricuspid area,* at the lower end of the sternum, (3) *pulmonary area,* at the inner end of the left second intercostal space, (4) *aortic area,* over the sternal end of the right second costal cartilage.

Inspection.—The following should be noted:—

1. *Shape of the praecordium.*—(*a*) *Abnormal bulging* apart from parietal causes indicates an enlargement of heart starting in early life.

A *fullness of the praecordium* may be due to pericardial effusion or rarely to mediastinal new-growths.

(*b*) A *retraction* is usually secondary to skeletal deformity of the thorax, either congenital, rachitic or occupational in origin. Rarely, it is due to chronic adhesive pericarditis and fibrosis of the lungs.

2. *Visible pulsations.* I. .*Over the praecordium.* (*a*) *Apical impulse.*—It is due to impact of the cardiac apex with the chest wall at each systole. It is the most reliable clinical evidence of the position of the apex of the heart. In the healthy adult, in the erect posture, it is seen over an area of about 1″ in diameter in the left fifth intercostal space just internal to the midclavicular line.

It is abnormally prominent in (i) thin chested people, (ii) overaction of the heart, due to excitement, exertion, fever, anæmia, thyrotoxicosis, neurocirculatory asthenia etc., (iii) hypertrophy of the ventricles (the lower sternum and the ribs may be visibly lifted), (iv) retraction of the left lung, (v) pericardial effusion, occasionally.

It may be invisible in (i) thick chested people, (ii) emphysema of the lungs, (iii) pericardial effusion, (iv) weak cardiac action.

(b) A pulsation in the left second and third intercostal space near the sternum.—May be due to (i) a dilated pulmonary artery, as in mitral stenosis, (ii) retraction of the left lung, as in pulmonary fibrosis, (iii) aneurysm at the commencement of the descending aorta.

(c) A systolic indrawing of the lower segment of the praecordium.—May be due to chronic adhesive pericarditis. A diffuse pulsation with systolic indrawing may also be seen in thin chested people due to systolic recession of the right ventricle from the chest wall.

II. *Pulsations outside the praecordium.* (*a*) *In the right second intercostal space near the manubrium,* a pulsation may be seen due to aneurysmal dilatation of the ascending aorta.

(*b*) *In the suprasternal notch,* may be seen in (i) aneurysm of the aortic arch, (ii) atheromatous dilatation of the aorta, (iii) aortic regurgitation, (iv) severe anæmia, (v) abnormal *thyroidea ima* artery or abnormal origin of the right subclavian artery.

(*c*) *Epigastric pulsation.*—This may be seen in (i) neurotic individuals with lax abdominal wall. The pulsation is systolic and transmitted from the abdominal aorta. (ii) excited action of the heart causing systolic retraction of the epigastrium, (iii) pulsation of the liver due to severe congestive cardiac failure, (iv) right ventricular enlargement, (v) aneurysm of the abdominal aorta.

(*d*) *Dilated pulsatile arteries in the intercostal spaces.*—May be seen in congenital coarctation of aorta, due to collateral circulation between the branches of the thoracic aorta with those of the subclavian artery, chiefly the scapular and internal mammary arteries.

(*e*) *Systolic retraction of the left lower intercostal spaces in the lateral or posterior chest wall* is seen in adhesive pericarditis. It is called *Broadbent's sign.*

Palpation.—For palpation, the physician stands or sits on the right side of the patient and places his right hand flat on the præcordium with fingers towards the apex of the heart. For detection of pulsations, at first the palm is used and then accurate localisation is made by the tips of the fingers.

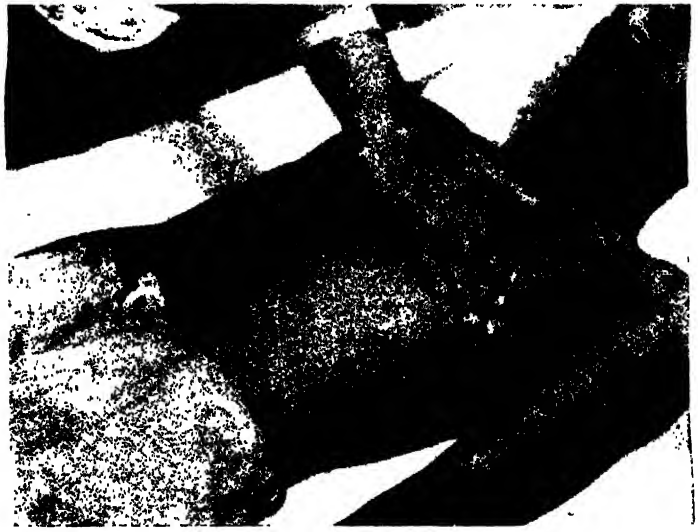


Fig. 2. Palpation of the apical impulse.

1. The findings of inspection as regards the shape of the præcordium and bulging or retraction elsewhere are confirmed by palpation.

2. *Pulsations.* I. *Apical impulse* is more accurately determined by palpation than by any other method. When palpating, the situation, character, rate and rhythm should be noted.

Situation.—When felt over a wide area, the outermost and the lowermost point, where a definite (not the maximal) thrust is felt by the finger tip, should be taken as the position of the apex of the heart. If a well defined thrust is not felt in the recumbent position, palpation should be done with the patient in the left lateral position. The site of apical impulse should be noted in relation to the intercostal spaces and to the left midclavicular line, and not to the mammary line which is variable in position. Normally, the apex is $\frac{1}{2}''$ internal to the midclavicular line in the left fifth intercostal space.

Normal variations.—(*a*) In children, it may be found in the left fourth space just outside the midclavicular line.

(*b*) In deep chested people, with high diaphragm, apex is found a little upwards and outwards.

(*c*) In the long narrow chested people, it is found more internally and in the left sixth space.

(d) In the left lateral position, the apex moves about 1″ outwards.

The apical impulse is found in abnormal situation in the following conditions:—

A. *Lateral displacement* of the heart and mediastinum by conditions in the lungs and pleural cavity. Thus, pleural effusion, pneumothorax, pulmonary newgrowths (with effusion), shift the heart to the opposite side.

In collapse and fibrosis of the lung, the apex of the heart is drawn to the same side.

B. *Enlargement of the heart.*—In left ventricular enlargement, the apex moves mainly downwards and in right ventricular enlargement, it moves mainly outwards.

C. *Upward displacement* to the third or fourth space may be due to (*a*) pericardial effusion, (*b*) raised intra-abdominal pressure from causes such as ascites, advanced pregnancy, marked splenomegaly, intra-abdominal tumours, (*c*) fibrosis of the upper lobe of the left lung, (*d*) paralysis of the left dome of diaphragm.

D. *In congenital dextrocardia,* apex-beat is found on the right side of the sternum.

The apical impulse may not be palpable due to the same causes which render it invisible. Before concluding that it is impalpable, congenital dextrocardia should be excluded.

Character.—Normally, the apical impulse is felt as a definite thrust of moderate strength and appreciable duration over a small area.

Variations in character are:—

A. Increased force and duration—A slow heaving impulse raising the chest wall indicates left ventricular hypertrophy.

B. A forcible but very short impulse—slapping in nature is characteristic of mitral stenosis. It may also occur in excitement, exertion or neuro-circulatory asthenia.

C. Weak or imperceptible impulse may be due to (*a*) thick chest wall, (*b*) emphysema of the lungs, (*c*) weak cardiac action, (*d*) pericardial effusion, (*e*) abnormal position or situation behind a rib.

D. A double apical impulse may be due to (*a*) bundle branch block in persons with normal blood pressure, (*b*) presystolic gallop rhythm in presence of hypertension.

Rhythm.—Irregularities, same as those seen in the pulse may occur in conditions like sinus arrhythmia, auricular fibrillation, extrasystole and heart-block. (See page 28).

II. *Other pulsations* in and outside the præcordium are looked for as in inspection. Their significance has already been mentioned.

3. *Thrills.*—A thrill is a tactile sense of vibration compared to the purring sensation felt when the hand is placed over the back of a cat. The following points should be noted about thrills.

(a) *Timing.*—Systolic or diastolic as determined by comparing with the apex-beat or the carotid pulse.

(*b*) *Site of maximum intensity.*

The common thrills found are

(*a*) In the mitral area—(i) a systolic thrill indicates mitral regurgitation; (ii) a presystolic or diastolic thrill indicates mitral stenosis.

(*b*) In the aortic area—(i) a systolic thrill indicates aortic stenosis, aortic aneurysm or dilated aorta; (ii) a diastolic thrill indicates a ruptured aortic valve and sometimes it is present in aortic incompetence.

(*c*) In the pulmonary area—a systolic thrill is found in congenital pulmonary stenosis.

(*d*) A little to the left of the pulmonary area—a more or less continuous or systolic thrill is found in patent ductus arteriosus.

(*e*) In the fourth intercostal space to the left of the sternum—a systolic thrill indicates patent interventricular septum.

(*f*) Thrill is also felt over arterio-venous aneurysms anywhere in the body.

Thrills are mostly pathognomonic of organic valvular disease, though occasionally systolic thrills may be present in severe anæmia or in forcible heart action of thin chested normal individuals.

4. *Pericardial friction rub* may be felt in fibrinous pericarditis. A friction rub can be distinguished from a thrill by being superficial, to-and-fro in character and being less constant in timing and situation.

Percussion.—Percussion is a method of physical examination in which a resonant note is produced by striking on surface of the body overlying an air-containing organ.

In examination of the heart, percussion is used to outline the area on the anterior chest wall overlying the heart, by finding out the limits between the resonant lung and the non-resonant heart. The heart being partially covered by the left lung, a partial resonance is obtained over the covered portion as elicited by light strokes. With firm percussion strokes however, the thin lung tissue between the heart and the chest wall cannot mask the dullness.

The outline of the cardiac dullness obtained by light percussion, is *superficial* or *absolute dullness,* and represents the uncovered portion of the heart. Its extent depends on the condition of the left lung or the left pleural cavity and its determination is of little importance in the diagnosis of heart disease.

The dullness obtained by moderately firm percussion is called *deep or relative dullness,* and this represents the actual anatomical outline of the heart. It is therefore, of greater clinical importance in determining enlargement of the heart or distension of pericardium by fluid. Unfortunately, however the method requires considerable practice and experience to reach any degree of accuracy. Even then, it is not possible to completely outline the heart by percussion. Its chief use is to make a rough estimate as to the position of the left border when the apex is not definitely palpable. The left and right borders cannot always be determined accurately. In thick chest-wall, emphysema of lung, left sided pneumothorax or in great distension of stomach, cardiac dullness may not be found at all.

In the routine examination, the following procedure is sufficient for all practical purposes.

1. The position of apical impulse is percussed.
2. Percussion of the left border in the third space.
3. Percussion of the right border and the cardio-hepatic angle.
4. Percussion of the second intercostal space from the left side across the sternum to the right.

Method.—The middle finger of the left hand is placed on the chest wall and its middle phalanx is struck by the middle finger of the right hand. The former is called the *pleximeter* and the latter *percussing finger.* The stroke is executed by a movement from the wrist and not from the elbow, and it may be light or firm according as superficial or deep resonance is to be elicited. Percussion should be done from the resonant to the dull area along a line perpendicular to the border percussed, the pleximeter finger being parallel to the border. The point where the note changes and a sense of resistance is felt, is noted.

Percussion of the apex.—A clear lung resonance is elicited in the fourth, fifth or occasionally the sixth space near the left axilla and then the border of the heart is approached, gradually moving the pleximeter finger inwards in the same space. If the dullness extends to the left beyond the point of palpable apical impulse, a *pericardial effusion* is present.

Percussion of the third space.—The third space on the left side is similarly percussed from the axilla inwards and the point of dullness noted. The distance of the left border from the middle line in the third space is normally about half the distance of the apex from the middle line. In right ventricular enlargement as in mitral stenosis or in dilatation of the pulmonary artery, dullness in the third space extends outwards, while the apex remains in the usual position.

Percussion of the right border.—For percussion of the right border, the liver dullness is to be determined first by percussing with moderate strokes in the midclavicular line from the second space downwards. This is found normally in the fifth space or

behind the fifth rib. Then the right border is percussed one space above the liver dullness, starting suffciently out on the right side and gradually approaching the heart. In the fourth or fifth space, the right border is found at a distance of 1 ¼" to 1 ½" from the middle line. It is not always possible to detect this border, specially in the presence of emphysema of the lung. A definite dullness beyond the normal limit indicates right auricular enlargement if displacement of the heart can be excluded.

The percussion of the cardio-hepatic angle is important in pericardial effusion when it becomes obtuse instead of the normal acute angle (*Rotch's sign*).

Percussion of the second intercostal space.—The second intercostal space is next percussed starting from the left side in the mammary line across the sternum to the right side. Normally, it is resonant throughout, the great vessels being deeply situated. This space may be dull in part or throughout in (*a*) aneurysmal dilatation of the aorta, (*b*) dilatation of the pulmonary artery, (*c*) displacement of the heart upwards, (*d*) patent ductus arteriosus.

If it is possible to outline the cardiac dullness, a characteristic shape may be seen in some conditions, such as, (*a*) a *boot-shaped* heart in left ventricular enlargement, (*b*) *globular* or *square-shaped* heart in right ventricular enlargement, (*c*) *pyriform* shape in pericardial effusion.

Auscultation.—In auscultation, we hear the cardiac sounds and any murmur or bruit. This can be done directly by placing the ear on the chest or indirectly by a *stethoscope*. The latter is universally used. The chestpiece is successively placed in the routine examination on the mitral, aortic, pulmonary and tricuspid areas. The first sound is best studied in the mitral area, and the second sound both in the aortic and pulmonary areas. The sounds should be studied first, and their character, intensity and duration are to be noted. During auscultation, it will be helpful to keep a finger on the apex-beat or the carotid pulse to time the sounds and murmurs.

First sound.—This is produced mostly by the myocardial contraction in ventricular systole and partly by closure of the auriculo-ventricular valves. It is best heard over the cardiac apex. Its character is best imitated by the word *lubb*.

Variations.—1. *Accentuation of intensity* may be due to: (*a*) thin chest wall, (*b*) overaction of the heart as in exertion, excitement, fever, anæmia, thyrotoxicosis, neurocirculatory asthenia, (*c*) hypertrophy of the ventricles—in left ventricular hypertrophy the sound is loud and prolonged, described as *booming*, (*d*) mitral stenosis—the sound is very short and sharp described as *clapping* or snappy.

2. *Weakness or indistinct first sound* may be due to: (*a*) thick chest wall, (*b*) emphysema of the lungs, (*c*) myocardial weakness from myocarditis, degeneration, fibrosis etc., (*d*) shock or collapse,

(*e*) general asthenia, as after prolonged illness, (*f*) pericardial effusion.

The first sound may be inaudible in the presence of loud apical systolic murmur, being masked by it.

3. *Reduplication.*—When the two ventricles contract asynchronously, the first sound may appear reduplicated as in bundle-branch block. Two separate sounds may also be audible in cases of auricular hypertrophy or in auriculo-ventricular conduction delay, the auricular contraction being audible.

4. *Shortness.*—Sometimes, the first sound becomes very short and sharp resembling the second sound. If this occurs with a very rapid heart rate, two similar sounds are audible at equal intervals, resembling the ticking of a clock. It is described as *tic-tac* or *foetal rhythm* because the fœtal heart sounds are normally of this character. It is an expression of exhaustion of the myocardium.

Second sound.—It is produced by the closure of the aortic and pulmonary valves at the beginning of diastole and by the consequent increased tension and vibration of the valves. It should be studied in the aortic and pulmonary areas. It can be best imitated by the word *dup.*

In the aortic area.—1. *Accentuation of the second sound* indicates: (*a*) high blood pressure, (*b*) aortic dilatation or aneurysm of the aorta. The sound in this condition has a metallic ringing quality simulating the sound produced on suddenly stretching a piece of linen (*Bruit d'Tabourke*).

2. *Weakness* of the second sound may be due to (*a*) low blood pressure as in collapse, (*b*) aortic stenosis, and (*c*) loud murmurs masking the second sound.

In the pulmonary area.—Accentuation occurs in: (a) excited action of the heart, (*b*) supine position, when the diaphragm is high, (*c*) conditions raising the pressure in the pulmonary circulation as mitral stenosis, left ventricular failure, emphysema, asthma, chronic bronchitis, fibrosis of lungs etc.

The second sound may be reduplicated either in the aortic or in the pulmonary area, due to asynchronous closure of the two semilunar valves. This is due to an abnormal increase in pressure in either the aorta or the pulmonary artery thus altering the normal relation between the two. The significance is the same as that of accentuation of the sounds.

Sometimes, *three heart-sounds* are audible. The third sound is almost always heard in diastole. This may occur in:—

1. *Normal hearts of children and adolescents.*—A physiological third sound due to the sudden opening snap of the auriculo-ventricular valves in early diastole. This is exaggerated in early mitral stenosis.

2. *Gallop rhythm.*—Three heart sounds occurring in a rapid heart simulate the footfalls of a galloping horse. The extra sound

may be audible in early diastole (*protodiastolic*), or in late diastole (*presystolic*). The sound is generally best heard at or just internal to the apex-beat. A gallop rhythm is most commonly associated with a dilated ventricle, usually the left, and is an indication of grave myocardial exhaustion. It is most commonly found in failing hypertensive heart, but may also be found in: (*a*) coronary thrombosis, (*b*) rheumatic carditis, (*c*) acute myocarditis of infective fevers like diphtheria, typhoid, influenza etc., (*d*) thyrotoxicosis, (*e*) severe anæmia etc.

The extra sound is believed to be produced by the impact of the inflowing blood from the auricles against the toneless wall of the dilated ventricles. A presystolic gallop may also be due to audible auricular contraction.

Adventitious sounds.—These are 1. cardiac murmurs, 2. cardio-respiratory murmurs and 3. pericardial friction rub.

Cardiac murmurs.—The mechanism of production of murmurs is not quite well understood. They are found whenever the normal relations between the cardiac chambers and their communicating orifices, are disturbed (as in stenosis, absolute or relative); or whenever there is a flow of blood in a direction opposite to the normal as in regurgitation through a valve. Their intensity depends on two factors,—(*a*) *the degree of change,* that is, the more severe the stenosis or regurgitation, the more intense is the murmur. (*b*) *the velocity of blood flow,* so that a doubtful murmur of early stenosis or regurgitation may be made more prominent by increasing the blood-flow by exercise.

When a murmur is audible, the following points are to be noted:—

1. The site of maximum intensity. 2. The relation to cardiac cycle, systolic or diastolic. 3. Character, such as, rough, rumbling, harsh, grating, soft, blowing or musical. 4. Intensity. 5. Propagation in a selective direction. 6. Constant or inconstant in different phases of respiration.

Murmurs in the mitral area.—1. *Systolic murmur* is found in:—

(*a*) Organic mitral regurgitation in which condition the valve cusps are structurally deformed, or too rigid to close the orifice completely. It is almost always of rheumatic origin.

(*b*) Functional regurgitation as a result of a dilatation of the muscular mitral ring from weakness or atony of the myocardium, the valve cusps being normal. This may occur in acute rheumatic carditis and toxæmias of infective fevers, in severe anæmia, coronary sclerosis, hypertension etc. These conditions are usually associated with left ventricular dilatation.

(*c*) Physiological, as in athletes or rapidly growing children without any evidence of cardiac enlargement, or organic heart disease.

The differences between the organic and functional mitral regurgitaton murmurs are shown in the following table.

TABLE II

	Organic.	Functional.
1. Character.	Harsh long and constant, loud.	Soft, blowing and musical, short.
2. Propagation.	Propagated to the left axilla.	No propagation.
3. Associated thrill.	Systolic thrill.	No thrill.
4. Previous history.	History of rheumatic infection present.	No history of rheumatic infection.
5. Cardiac enlargement.	Present.	May be absent.
6. Associated mitral stenosis.	May be present.	Absent.

It must be mentioned that the organic and functional murmurs may resemble each other in their character and propagation. The only reliable guides are a definite history of rheumatic infection, and a definite evidence of mitral stenosis, which must be looked for with all possible care.

A systolic murmur at the apex must be distinguished from a cardio-respiratory murmur and a pericardial friction rub. The former is produced by sudden suction of air into the lung alveoli around the heart by the partial vacuum, created at each systole. Such a murmur is soft, short and superficial, and heard best in inspiration.

A pericardial friction rub is recognised by its superficial, rubbing, to-and-fro character. It is inconstant in time and position.

A systolic murmur at the mitral area may also be transmitted from other areas, when its point of maximum intensity will be elsewhere.

2. *Diastolic murmur in the mitral area* is heard in: (*a*) mitral stenosis, (*b*) aortic regurgitation, being transmitted from the aortic area, (*c*) relative mitral stenosis—Austin Flint murmur.

(*a*) *The murmur of mitral stenosis.—Character.*—It is rough, rumbling and of low pitch. It has a marked presystolic accentuation. The so-called crescendo character is due to its sharp ending in an accentuated first sound.

Point of maximum intensity.—Over a small area at the apex.

Propagation.—It is very localised and has no selective propagation.

How elicited.—It is best heard in the recumbent posture and best in the left lateral position. In doubtful cases, auscultation should be done immediately after some exercise and with the patient in the left lateral position, when the murmur will be accentuated.

Mechanism of production and variation of murmur with the stages of stenosis.—The murmur is due to passage of blood through the stenosed mitral orifice during diastole. Its intensity depends on the degree of stenosis and the velocity of blood-flow. In advanced stages, the murmur occupies the full diastole, with accentuation in early diastole and presystole, due to rapid blood-flow from a suction action of the relaxing ventricles in early diastole, and auricular contraction in presystole. In early stages, the murmur may be only presystolic, or more often early diastolic, especially in young children. When auricular systole is absent due to auricular fibrillation, the presystolic accentuation disappears.

(*b*) In aortic regurgitation, an early diastolic murmur may be heard at the mitral area. It is soft blowing in character and has no presystolic accentuation. Its point of mixiumum intensity is in the third space near the sternum.

(*c*) *Austin Flint murmur.*—This is a presystolic murmur heard in some cases of aortic regurgitation without any organic stenosis of mitral orifice. It is believed to be due to a relative mitral stenosis.

Murmurs in the aortic area.—1. *Systolic murmur.* This may be due to: (*a*) dilatation of the aorta from—(i) syphilitic aortitis, (ii) atheroma, (iii) severe anæmia with atony, (*b*) Aneurysm of the ascending aorta, (*c*) Aortic stenosis, (*d*) Transmitted murmur from the pulmonary area.

The first two conditions produce murmur by causing a relative or functional stenosis without organic change in the aortic valves. The third condition is associated with organic change in the cusps. The distinction between functional and organic stenosis murmurs is shown in Table III.

2. *Diastolic murmur.*—This is found in: (*a*) aortic regurgitation due to rheumatic infection, syphilis or atheroma; (*b*) torn aortic valves, as in ulcerative endocarditis.

Aortic regurgitation murmur.—Character.—Soft and blowing Begins with maximum intensity immediately after the aortic second sound and gradually falls off. The duration depends on the degree of regurgitation.

Point of maximum intensity.—Third and fourth intercostal space at the left border of the sternum or in the pulmonary area.

Propagation.—Downwards along the left margin of the sternum.

How elicited.—It is best heard in the erect posture. In doubtful cases, patient is asked to hold his breath at the end of expira-

tion, when the murmur will be better heard. Direct auscultation with ear placed on the chest wall also helps in doubtful cases.

TABLE III

	Organic.	Functional.
1. Character.	Rough, loud, may be grating.	Soft and blowing, of low intensity.
2. Propagation.	Upwards over the large vessels of the neck. Sometimes to the apex also.	Not widely propagated.
3. Thrill.	Systolic thrill over aortic area.	No thrill.
4. Aortic second sound.	Diminished or absent.	Accutuated and ringing.
5. Pulse.	High tension, slowly rising sustained pulse.	No characteristic pulse.

Both systolic and diastolic murmurs at the aortic area may be simulated by pericardial friction rub, which is usually of a superficial to-and-fro character.

Murmurs in the pulmonary area. 1. *Systolic murmur.*—This is the commonest of all murmurs heard over the heart. The causes are:—

(*a*) Relative pulmonary stenosis, due to dilatation of the pulmonary artery, whose wall being thinner and less elastic than that of the aorta, is easily dilatable in such conditions as: (i) anæmia and febrile states with lack of general tone, (ii) conditions of increased resistance in the pulmonary circulation, as in emphysema, asthma, chronic bronchitis, fibrosis of the lung etc., (iii) mitral stenosis and left ventricular failure impeding venous return from the lung, (iv) a supine position with a raised diaphragm, and forced expiration.

(*b*) An organic stenosis of congenital origin is a less common cause of pulmonary systolic murmur, which is then harsh and loud, and associated with a thrill.

(*c*) Patent ductus arteriosus, aneurysm of the descending aorta and coarctation of aorta are rare causes of systolic murmur in the pulmonary area.

(*d*) It may be a transmitted murmur from other areas.

2. *Diastolic murmur.*—The causes are (*a*) Aortic regurgitation.

(*b*) Pulmonary regurgitation, which is usually functional due to

dilatation of the pulmonary ring along with a dilatation of the pulmonary artery. When this happens in mitral stenosis, the pulmonary diastolic murmur is called *Graham Steell murmur.*

Murmurs in both the above conditions are similar in character and and propagation. Aortic regurgitation is distinguished by a water-hammer pulse, and pulmonary regurgitation by an exaggerated pulsation in the root of lungs as seen by fluoroscopy. The murmur in the latter condition may also be heard at the lung bases.

Murmurs in the tricuspid area.—In very rare cases of tricuspid stenosis, a presystolic murmur, of the same character as that of mitral stenosis, is heard.

Although functional tricuspid regurgitation presumably occurs quite frequently in right ventricular failure due to dilatation of the right ventricle and consequently of the tricuspid ring, a systolic murmur is very rarely heard.

Other murmurs. (1) *Roger's murmur.*—A systolic murmur with maximum intensity at the left third and fourth intercostal space near the sternum is heard in patent interventricular septum.

(2) *Machinery or humming top murmur.*—A more or less continuous, loud, noisy murmur with systolic or diastolic accentuation is heard in the left second intercostal space just outside the pulmonary area in patent ductus arteriosus. It is described as machinery or humming top murmur because of its noisy character.

(3) *Vascular murmurs.*—(*a*) *Duroziez's murmur*—a diastolic murmur over the femoral artery in aortic regurgitation.

(*b*) Systolic murmur over aneurysms.

Cardio-respiratory murmurs.—In each cardiac systole a partial vacuum is produced in the overlying lung and a sound may be produced by a sudden entry of air into the alveoli. This may be mistaken for a systolic intracardiac murmur. Similarly, in diastole, the expanding ventricles may force some air out of the overlying alveoli and cause a diastolic murmur. Such murmurs are heard best at or near the apex. They are short and superficial, and are heard better, or only, during inspiration.

Pericardial friction rub.—In fibrinous pericarditis, a rough superficial to-and-fro sound is audible specially at the base of the heart. It varies from time to time, and its relation to the cardiac systole and diastole, is less constant.

Pulse.—For the routine clinical examination, the radial pulse is felt as it is most superficial and easily accessible. The middle three fingers of the right hand are placed lightly on the artery and are gradually pressed until a maximum pulsation is felt. The degree of pressure necessary for this, indicates the *tension,* so that maximum pulsation with a very light touch, indicates a low tension.

The size of the pulse and the amplitude of pulsation indicate its volume.

The following points must be noted about the pulse: 1. Rate. 2. Rhythm. 3. Tension. 4. Volume. 5. Condition of the arterial wall.

I. *Rate.*—It varies according to age, sex and activity; the normal average being 72 per minute. It may be as high as 90 or as slow as 60 per minute in young healthy adults.

Tachycardia.—It means abnormal increase of pulse-rate.

Causes. 1. *Simple or sinus tachycardia.*—The cardiac rate is increased but the impulse for cardiac contraction arises at the normal pace-maker, the sino-auricular node. The rate rarely exceeds 160 per minute; and it responds to exercise and rest by gradual increase and decrease.

It may occur in (*a*) exercise and emotional disturbances, (*b*) febrile states, (*c*) active tuberculosis or rheumatic infection, (*d*) heart failure, (*e*) anæmia and hæmorrhage, (*f*) shock and collapse, (*g*) thyrotoxicosis, (*h*) neurocirculatory asthenia, (*i*) drugs such as atropine (by depressing the vagus), adrenaline, ephedrine, excess of tobacco, coffee etc., or caffeine by stimulating the sympathetic.

2. *Auricular fibrillation and flutter.*—The cardiac impulse arises from an abnormal circus movement in the auricles and not from the pace-maker (S-A node). In fibrillation the rate is 90 to 160 per minute and the pulse is irregularly irregular. Exercise increases the rate and irregularity of the pulse. In flutter, the rate is 140 to 180 per minute, pulse is regular, and it changes suddenly by multiples on exercise or is not affected at all. It is temporarily slowed by pressure on the carotid sinus.

3. *Paroxysmal tachycardia.*—It is a sudden onset and offset of tachycardia due to no apparent exciting cause; the impulse arising outside the normal pace-maker. Pulse rate varies from 120 to 220 per minute and is not affected by exercise, rest or change of posture. Pressure on carotid sinus (reflex vagus stimulation) has no effect on the rate, or if any effect at all, the tachycardia ceases suddenly.

Bradycardia.—It means abnormal slowing of the pulse-rate.

1. *Simple or sinus bradycardia.*—The cardiac impulse arises at the normal pace-maker and the ventricles respond to each impulse but the rate is very slow. The rate rarely falls below 50 per minute and it responds to exercise by gradual increase.

It occurs in (*a*) Sleep and rest. (*b*) Increased intracranial pressure. (*c*) Obstructive jaundice. (*d*) Myxœdema. (*e*) Vagal stimulation (i) reflexly, as in vasovagal syncope, (ii) by drugs as digitalis. (*f*) Convalescence from acute illness. (*g*) Certain infective fevers (the rate is relatively slow) as typhoid fever, mumps, dengue, influenza.

2. *Heart-block.*—The ventricles fail to respond to some of the impulses arising at the pacemaker (*incomplete heart-block*), or to

all the impulses (*complete heart-block*) and assume an independent rhythm. In incomplete heart-block with 2:1 rhythm, pulse rate is regular and between 40 to 50 per minute. Rate may be abruptly doubled on exercise or inhalation of amyl nitrite. In complete heart-block, rate is generally below 36 per minute and is not affected by exercise or amyl nitrite inhalation.

II. *Rhythm.*—Irregularities of pulse may be present with or without a dominant rhythm.

1. *With dominant rhythm.* (*a*) Sinus arrhythmia—alternate acceleration and slowing with the phases of respiration.

(*b*) Extra systole—causing dropped beats or coupling or tripling of beats.

(*c*) Incomplete heart-block—causing dropped beats or coupling or tripling.

2. *Without dominant rhythm.*—Completely irregular pulse in which no two beats are similar, either in spacing or strength, as in auricular fibrillation.

III. *Tension.*—High tension occurs in all conditions giving rise to high blood pressure, as in essential hypertension, acute or chronic nephritis, aortic stenosis, thyrotoxicosis etc.

Low tension is found in (*a*) shock, (*b*) lack of arteriolar tone, (c) rundown, anæmic or debilitated states or (*d*) Addison's disease.

IV. *Volume.*—It depends on the cardiac output. In hæmorrhages or dehydration, or in low venous return to the heart from peripheral vascular stasis, pulse is of very low volume and tension.

Inequality of the pulses in the two wrists may be due to (*a*) aneurysm of the aorta, (*b*) mediastinal growths, (*c*) abnormalities in the arteries.

An increase in the volume of the pulse may normally occur in inspiration and a decrease in expiration. When abnormal decrease, almost to disappearance, occurs in inspiration, it is called *pulsus paradoxus.* This occurs in pericardial effusion or constrictive pericarditis.

Alternate big and small beats occur in *pulsus alternans,* which is an indication of extreme myocardial exhaustion.

V. *Condition of the arterial wall.*—The artery is completely emptied by pressure of the fingers and it is rolled between the fingers and the underlying bone. In thickening and hardening of the artery due to arteriosclerosis it is felt like a cord. Irregularity and tortuosity should also be noted.

All the superficial arteries are to be examined, such as, the radial, brachial, temporal and the dorsalis pedis arteries.

A graphic record of the arterial pulse (sphygmogram) and of the venous pulse (phlebogram) over the neck veins is a very useful method of investigation. Simultaneous tracing of the venous and radial pulsation can be recorded by an instrument called *polygraph.*

Special types of pulse. 1. *Corrigan's or water-hammer pulse (collapsing pulse).*—A sudden forcible upstroke followed by an immediate fall; the artery collapsing to an almost empty condition in diastole. It is best appreciated when the wrist is grasped by the

Fig. 3. Normal radial sphygmogram.

palm and raised above the patient's head, thus exaggerating the diastolic collapse; a distinct sharp impact is felt by the fingers, whereas with a normal pulse no pulsation is felt.

Causes.—(a) Aortic regurgitation. (b) Severe anæmia. (c) Thyrotoxicosis. (d) Nervous excitement. (e) Arterio-venous aneurysm. (f) Patent ductus arteriosus. (g) Occasionally in high blood pressure with high pulse pressure.

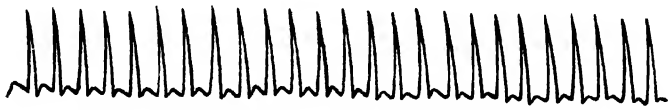


Fig. 4. Sphygmogram showing water-hammer pulse.

2. *Pulsus tardus or plateau-pulse.*—It means a slow gradually rising sustained pulse. It is seen in aortic stenosis or in conditions of hypertension.

3. *Anacrotic pulse and pulsus bisferiens.*—This is like the pulsus tardus, but with a double wave on the upstroke. This is also found in aortic stenosis.

Fig. 5. Radial sphygmogram showing anacrotic pulse.

4. *Dicrotic pulse.*—There is a double wave on the down stroke. It is seen in conditions of low diastolic pressure with overaction of the heart, as in febrile states, particularly typhoid fever.

5. *High bounding pulse.*—A rapid pulse of large volume but low tension is seen in acute infective fevers with excited cardiac action and peripheral vasodilatation, as in pneumonia.

6. *Thready running pulse.*—A rapid pulse of low volume and tension. It is seen in peripheral failure, shock or hæmorrhage. In extreme cases, pulse becomes imperceptible.

Auscultation over large arteries like the femoral is sometimes done. In aortic regurgitation, a systolic pistol-shot sound due to a sudden entry of blood into a collapsed empty vessel, and a diastolic murmur, the Duroziez's murmur, are heard over the femoral artery.

Lastly, capillary pulsation in the peripheries like the finger tips or the lips is looked for. When exaggerated, capillary pulsation is seen as alternate flushing and blanching if a glass slide is lightly pressed on the lip. This is characteristic of aortic regurgitation.

Blood pressure.—Blood pressure can be accurately measured by a *sphygmomanometer*. Several types of instruments are in use, of which, the one most commonly used, is a modification of the Riva Rocci's mercury sphygmomanometer. It consists of a manometer graduated in millimeters, a mercury reservoir, which is connected with manometer and a pneumatic rubber bag (12 cm. in width) to be tied around the arm. The bag can be inflated by a rubber pump. The pressure in the bag is recorded by the column of mercury in manometer.

Fig. 6. Measurement of blood pressure.

Method of use.—The patient should be in a position of comfort and ease, lying in bed on his back, with the arm in comfortable position at the level of the heart. The pneumatic bag is tied loosely around the arm well above the elbow, the bag being on the medial side of the arm. The outlet screw of the pump is closed; the fingers of the left hand are placed on the radial pulse and the pressure in the bag is raised by working the pump with the right hand until the radial pulse is no longer palpable. The chestpiece of a stethoscope

is now placed on the brachial artery at the cubital fossa and the pressure is gradually lowered by loosening the outlet screw of the pump. The sounds heard over the artery undergo a series of changes in which five stages are recognised with the lowering of pressure. The stages are:—

1. Initial loud and clear sounds. 2. Sounds muffled with the character of murmur. 3. Loud and clear sounds. 4. Dull sounds. 5. Cessation.

The reading in the manometer is noted at the beginning of the first stage, which gives the *systolic pressure,* and at the beginning of the fourth stage, which gives the *diastolic pressure.*

Sometimes in the second stage, the sounds disappear completely, to reappear in the third stage. This is called the *auscultatory gap.* It most often occurs in high blood pressure. The systolic pressure may then be wrongly recorded at the beginning of the third stage unless the pressure has been raised sufficiently high to obliterate the radial pulse. This error may also be avoided if a preliminary observation of the systolic pressure is made by palpation of the radial pulse, that is noting the pressure at which the pulse reappears after being obliterated by raising the pressure in the bag sufficiently high.

The pressure in healthy people varies with age, sex, physical activity, mental excitement etc. A systolic pressure above 150 mm. and a diastolic above 90 mm. at any age are regarded as high pressure; and a systolic pressure below 90 mm. and diastolic below 50 mm. are regarded as low blood pressure.

Exercise tolerance test.—In cases of doubtful cardiac efficiency, it becomes necessary to study the reaction of the heart to exercise. Obviously it is unnecessary in the presence of symptoms and signs of cardiac failure. Two tests are in use.

1. *Simple tests.*—(a) Walking briskly up a flight of forty steps. (*b*) Hopping twenty times on the left and twenty times on the right foot. (*c*) Stepping on and from a stool 18 inches high, twenty times.

2. *Strenuous test.*—It consists in lifting heavy weights from the floor above the head.

The pulse is counted immediately before and after the exercise and again after a rest of two minutes. A person with normal cardiac reserve feels no discomfort or dyspnœa after the exercise. The pulse does not increase more than ten to twenty beats per minute and it reaches the previous rate within two minutes of rest.

Examination of other systems. I. *Respiratory system.—Subjective symptoms.*—There may be: (a) cough and expectoration due to œdema of lungs in left ventricular failure, (*b*) hæmoptysis due to pulmonary congestion in mitral stenosis, (*c*) acute paroxysmal dyspnœa due to early left ventricular failure, (*d*) acute pain in the chest due to pulmonary infarction.

Objective signs.—Lung and pleura should be examined by inspection, palpation, percussion and auscultation for evidences of (*a*) œdema at the bases, and (*b*) hydrothorax.

II. *Alimentary system.—Subjective symptoms.*—Anorexia, flatulence and right hypochondriac pain may be present due to passive congestion of liver and gastro-intestinal mucous membrane.

Objective signs.—The most important examination is for evidence of enlargement of liver. In right ventricular failure, the liver is enlarged and tender; it is pulsatile also in cases with tricuspid regurgitation.

Next is the evidence of ascites which also occurs in right ventricular failure.

III. *Nervous system. Subjective symptoms.*—These may occur due to deficient cerebral circulation in cardiac failure, such as, headache, insomnia, failure of memory, inability to concentrate, giddiness or fainting attacks, epileptic fits, confusion, delirium etc.

Objective signs.—In syphilitic heart disease, neuro-syphilis may be co-existent, so that changes in the knee jerks and pupils are to be particularly looked for. In essential hypertension or arteriosclerotic heart disease also, cerebral vascular lesions may co-exist or complicate, when hemiplegia is present.

IV. *Urinary system. Subjective symptoms.*—(*a*) Scanty urine due to passive congestion of kidneys in right sided failure. (*b*) Polyuria, specially at night, due to nephrosclerosis in hypertension.

Objective signs.—Changes in the urine such as albuminuria and casts in the urine indicating changes in the kidney secondary to heart failure or hypertension.

V. *Examination of the fundus of the eye.*—An electric ophthalmoscope is most useful for the purpose. The pupils should be dilated before hand with a few drops of ½% solution of homatropine.

Important findings in heart cases are,—

(*a*) *Evidence of arteriosclerosis.*—The arteries are tortuous and shiny, described as having cork-screw, silver wire, or copper wire appearance. The veins are compressed where they cross the arteries.

(*b*) *Hypertensive neuro-retinopathy.*—The optic disc is swollen with exudate. The margin is indefinite and cloudy. Hæmorrhages may be present. The retina shows patches of white exudate and areas of hæmorrhage. At the macula, a star-shaped figure may be seen due to white streaks of exudate.

This is seen in essential hypertension (malignant type) or in chronic nephritis. It is of very bad prognosis.

(*c*) *Arteriosclerotic retinopathy.*—Besides evidences of arteriosclerosis of the retinal vessels, there are areas of hæmorrhages and white patches in the retina, but unlike neuro-retinopathy, *the optic disc is unaffected.* This condition is usually seen in benign hypertension. (See frontispiece.)

Special investigations.—1. *Study of the cardiac silhouette as obtained by X-rays.*—The changes in the different chambers of the heart or in the aorta, may alter the outline of the heart in different positions in characteristic ways, and thus help in the diagnosis. Moreover, X-rays provide the most reliable method of determining the cardiac size by accurate measurement of different axes.

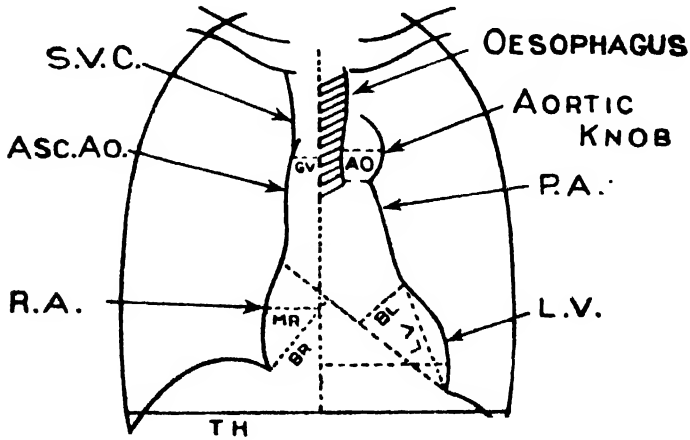


Fig. 7. Outline of heart and big vessels as seen under X-rays. S.V.C.—Superior vena cava. Asc. Ao.—Ascending aorta. R.A.—Right auricle. T.H.—Diameter of the thorax. P.A.—Pulmonary artery. L.V.—Left ventricle.

2. *Electrocardiography.*—This is a graphic record of the electrical changes, which accompany the origin and spread of the cardiac impulse through the myocardium during the auricular and ventricular systoles. It is of great help in diagnosis of cardiac arrhythmias and coronary diseases. (See Appendix A.).

3. *Circulation time.*—This is estimated to differentiate between cardiac and pulmonary dyspnœa, and in the diagnosis of left or right ventricular failure.

Arm to tongue circulation time.—This is estimated by injecting either 5 c.c. of 20% solution of Decholin (sodium dehydrocholate), or 6 c.c. of 10% solution of magnesium sulphate quickly through a wide bore needle into the antecubital vein and noting the time when the patient can feel a bitter taste (with Decholin), or a hot sensation (with magnesium sulphate) in the tongue. Normally, the time is on an average 13 seconds.

Arm to lung circulation time.—This is estimated in the same way as above by injecting 5 minims of ether in 5 minims of normal

saline and noting the time when a smell of ether in the breath is felt. Normal average time is 5·7 seconds. This indicates the functional efficiency of the right ventricle, and the difference between this and the arm to tongue time indicates the effciency of the left ventricle. In isolated left ventricular failure arm to tongue circulation time is prolonged, whereas the arm to lung time is normal. In chronic bronchitis, emphysema or asthma without heart-failure, circulation time is normal.

CHAPTER II

CIRCULATORY FAILURE

Circulatory failure generally means an inadequate supply of oxygenated blood to the tissues due to inefficient circulation. This may arise in two ways, *viz.*, due to an inefficiency of the myocardium in pumping out the requisite amount of blood, or due to a reduction in the volume of blood in effective circulation. Circulatory failure, therefore, is of two varieties,—1. cardiac failure, 2. general vascular failure or peripheral failure.

Cardiac Failure

Cardiac failure is an inability of the heart to maintain an efficient circulation through the body, either on exertion or at rest. This may be manifested in two ways.

1. *Failure with congestion.*—The heart is unable to empty itself completely and therefore congestion develops in the auricles and the veins behind.

2. *Failure with ischaemia of the vital organs and tissues.*—(*a*) Ischæmia of the brain—*Syncopal failure.* (*b*) Ischæmia of coronary circulation—*Anginal failure.*

CONGESTIVE CARDIAC FAILURE

The chief manifestation of this type of failure, namely, passive venous congestion behind the failing chamber, is due primarily to inability of the ventricles to discharge their contents completely into the arterial system. The result is distension of the ventricular cavity and an impediment to venous return. Consequently, stasis occurs in the auricles and the big veins opening into them. A rise of venous pressure and passive congestion of organs follow.

The causes of this congestion or incomplete emptying of the ventricles may be inefficient contraction of the myocardium or abnormal increase in the resistance to emptying of the ventricles: such as high blood pressure or valvular stenosis. The normal response of a healthy myocardium to an increase of resistance is hypertrophy, which is generally sufficient to overcome the resistance, unless it is sudden and severe as in pulmonary embolism with acute dilatation of the heart. Mere increase of resistance never reaches such severity as to lead to heart failure, unless the myocardial efficiency is impaired by infection, inflammation, coronary narrowing or fatigue associated with rapid ventricular rate. The condition of the myocardium is the deciding factor in the onset of heart failure irrespective of the burden on it. The inefficiency of the myocardium may arise from:—

1. Organic diseases, like rheumatic carditis, coronary thrombosis, ischæmic fibrosis from coronary atheroma, acute myocarditis of infective fevers etc.

2. Deficient nourishment due to coronary narrowing, severe anæmia etc.

3. Overwork and fatigue due to thyrotoxicosis, paroxysmal tachycardia, auricular fibrillation, prolonged hypertension, etc.

Presence of an increased resistance may however hasten or aggravate the failure and determine which ventricle will fail first or predominantly.

Congestive cardiac failure may be (*a*) left ventricular, (*b*) right ventricular, and (*c*) mixed.

Left ventricular failure. *Common causes.*—(a) Hypertension, (*b*) Coronary disease, (*c*) Aortic regurgitation or stenosis, (*d*) Thyrotoxicosis.

Effects.—(*a*) Left ventricular and left auricular dilatation (*b*) congestion of pulmonary circulation, at first during exertion, later at rest. (*c*) Dilatation of pulmonary artery and pulmonary conus of the right ventricle. (*d*) Increased work for the right ventricle which in its turn fails sooner or later.

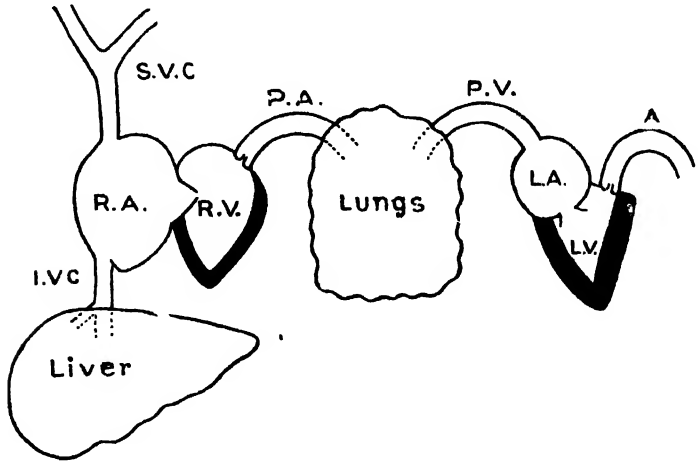


Fig. 8. The effect of right ventricular and left ventricular failure. In right ventricular failure, congestion and backpressure affect the right auricle, sup. vena cava, inf. vena cava, the liver and the systemic veins; in left ventricular failure, the left auricle and the lungs are similarly affected. R.V.—Right ventricle. L.V.—Left ventricle. R.A.—Right auricle. L.A.—Left auricle. P.A.—Pulmonary artery. P.V.—Pulmonary vein. A.—Aorta. S.V.C.—Superior vena cava. I.V.C.—Inferior vena cava.

Signs and symptoms.—1. Dyspnœa on exertion. 2. Cardiac asthma. 3. Apex beat shifted downwards and outwards. 4. A soft systolic murmur at the apex may appear. 5. Moist sounds at the lung bases. 6. Absence of signs of systemic venous congestion *viz.*, engorged neck veins, enlarged and tender liver or dependent œdema. 7. Gallop rhythm at the apex may be present in severe cases. 8. Pulmonary second sound is accentuated. 9. Pulsus alternans may be present. 10. Left sided hydrothorax. 11. Prolongation of arm to tongue circulation time, while arm to lung time remains normal

Right ventricular failure. *Causes.*—1. Mitral stenosis. 2. Secondary to left ventricular failure. 3. Pulmonary diseases like emphysema, asthma, chronic bronchitis, fibrosis etc. 4. Congenital pulmonary stenosis.

The myocardial changes of rheumatic infection which accompany mitral stenosis, the associated coronary changes accompanying causes of left ventricular failure and the anoxœmia accompanying the chronic pulmonary diseases, affect the ventricular myocardium primarily; the increased strain imposed on the right ventricle in the above conditions, precipitates its failure.

Effects.—Right ventricular and auricular dilatation and congestion in the superior and inferior vena cavæ, the former leads to engorgement in the neck veins, and the latter to a venous stasis in the hepatic veins with consequent passive venous congestion of the liver. The general increase of venous pressure in the systemic, as well as the portal circulation occurs. In the dependent parts of the body, gravity adds to the venous stasis and œdema results.

Signs and symptoms.—1. Dyspnœa on exertion or at rest also occurs in right ventricular failure, as most of the causes leading to it produce pulmonary congestion as well. Sometimes however, the dyspnœa may actually diminish when the right ventricle fails as in some cases of left ventricular failure; the cardiac asthma of earlier stages disappears when venous congestion and œdema develop due to right ventricular failure

2. Cyanosis, due to venous stasis.

3. Enlargement of the heart more in its transverse diameter than in the long axis. Both the right border and the left border in the third space move outwards.

4. Engorged neck veins, which may pulsate.

5. Enlarged, tender, and sometimes pulsatile liver.

6. Pitting œdema of dependent parts.

7. Arrhythmia, specially auricular fibrillation is common.

8. A gallop rhythm near the lower end of the sternum in severe cases.

9. Ascites and in severe cases a hydrothorax, usually right-sided.

10. Prolongation of both arm to tongue and arm to lung circulation time.

Diagnosis.—In the early stages, a definite limitation of tolerance to exercise and signs of organic heart disease, either clinical or electrocardiographic, are sufficient for diagnosis. Later, when congestion develops, a combination of cervical venous engorgement and enlarged and tender liver, or of dyspnœa and pulmonary œdema, associated with signs of organic heart disease and cardiac enlargement, would be diagnostic. A patient with œdema of dependent parts must have his neck veins engorged and liver enlarged and tender, if œdema is of cardiac origin. (See also the differential diagnosis of œdema, in Part III).

Effort syndrome or neurocirculatory asthenia must be distinguished by absence of cardiac signs, and relation of the dyspnœa more to emotion than effort.

Course and prognosis.—The immediate prognosis depends on the degree of failure as judged by the severity of reduction of cardiac reserve, the degree of venous engorgement and severity of œdema. Death in the first attack is rare. Sufficient improvement with disappearance of venous engorgement usually results on treatment. The ultimate prognosis, however, depends on the cause of failure. If the organic change in the myocardium and its cause are recoverable, then prognosis is good, otherwise the expectation of life must be limited.

Secondary infections may terminate life at any stage of chronic congestive failure.

Treatment.—The general principles of treatment are

1. *Rest* to the myocardium by: (*a*) physical and mental rest if necessary with sedatives, (*b*) reduction of ventricular rate with digitalis and allied drugs, (*c*) reduction of strain on the heart as far as possible, such as by reduction of high blood pressure, removal of œdema, reduction of venous engorgement by venesection and reduction of overweight in obesity.

2. *Supply of adequate nourishment and oxygen* to the myocardium by: (*a*) oxygen inhalation, (*b*) improvement of coronary blood flow, when coronary arteries are narrow, by theophyllin-ethylene-diamine (Aminophylline), (*c*) correction of anæmia, (*d*) glucose by mouth or intravenously.

3. *Symptomatic relief.*

The patient is put to absolute bed-rest in a propped up position, which gives him comfort. General hygienic measures are taken, such as cleansing the mouth, sponging, attention to skin at pressure points to prevent bedsore by rubbing regularly with spirit, drying and powdering etc.

Oxygen inhalation preferably by a B.L.B. mask, or by a nasal catheter at the rate of 120 bubbles per minute is given continuously.

Digitalis should be given in either of the three forms,—(*a*) powdered leaf, (*b*) tincture of digitalis and (*c*) Digoxin (a glusoside isolated from digitalis lanata). With the former two, full thera-

peutic effect is slow, because it requires accumulation in the body of a certain minimum amount. As the excretion is slower than absorption, small doses given over several days, cause sufficient concentration. In mild cases, tincture digitalis 20 minims (equivalent to 2 grains of powdered leaf) every 6 hours is given until the desired effect is obtained, which is usual in three to four days. Then a maintenance dose of 20 to 30 minims is given daily to replace the daily excretion. Large doses may be given at once to have immediate full effect, but this is unsafe because of gastric irritation and individual susceptibility unless the patient is under constant control and supervision. An initial dose of one and a half drachm of tincture digitalis is given; six hours later, if there is no slowing of pulse, another dose of one drachm is given, and a further dose of half drachm six hours later, if there be no slowing still. If, however, slowing of pulse is seen, 15 to 20 minims is given every six hours until the desired effect (i.e., the pulse rate falling to 60 to 70 per minute) is attained, or until toxic symptoms appear.

Digoxin given by mouth acts much more quickly than digitalis. A dose of 1 to 1·5 mgm is given every 6 hours until the desired effect is produced. For maintenance of effect, 0·25 mgm twice a day is sufficient.

During digitalis administration, constant watch should be kept on ventricular rate for abnormal slowing or appearance of extra-systoles, when the drug should be stopped. Nausea, vomiting, headache etc., are mild toxic symptoms which may also require discontinuance of the drug.

Symptomatic treatment.—Severe venous engorgement and *hepatic congestion* require venesection and 6 to 8 ounces of blood-letting or application of 6 to 12 leeches over the right hypochondrium.

Paroxysmal nocturnal dyspnoea or *cardiac asthma* requires sedatives. In mild cases, codeine phosphate gr. ½ with phenobarbitone or luminal gr. 1 may be given. In severe cases, morphine sulphate gr. $\frac{1}{6}$ to ¼ should be given subcutaneously. If œdema of lungs is present, atropine sulphate gr. 1/100 should also be given to counteract the respiratory depression of morphine.

Cyanosis should be treated with oxygen inhalation.

Œdema.—Reduction of fluids and sodium salts. Saline purgatives to ensure one fluid motion a day. Diuretics are given either as diuretin gr. 10 t.d.s., or in severe and obstinate cases, mercurial diuretics, with proper precautions to exclude impairment of renal function. After a preliminary course of ammonium chloride gr. 20 t.d.s., for two days, ½ c.c. of mersalyl (Salyrgan) is given intramuscularly to test for idiosyncrasy. If this is absent, 2 c.c. intramuscularly is given.

Insomnia.—Barbiturates, bromides and chloral hydrate, morphine etc., can be used.

Diet.—In the stage of congestion simple easily digestible food in small amounts at a time, should be given, such as milk, biscuits, bread and butter, fruit juice etc. A total of 800 to 1,000 calories in 24 hours is sufficient. With improvement, diet is gradually increased, but overfeeding and bulky meals must be avoided. Total fluids should not exceed 40 ounces in twentyfour hours.

General management.—The patient should be kept in bed until all the signs of congestion have disappeared. Then after allowing sufficient time for recovery of cardiac reserve, walking exercise within the limit of cardiac tolerance should gradually be allowed.

SYNCOPAL FAILURE

Syncope is a temporary loss of consciousness due to cerebral anæmia. This may be of vascular or cardiac origin as we have stated before. (See page 9).

Cardiac syncope, that is, when the cerebral anæmia is directly due to inadequate action of the heart, is seen in:—

1. Heart-block—(*a*) when the ventricular rate falls below 20 per minute in complete block;

(*b*) when temporary cardiac arrest occurs in complete or incomplete block.

2. Rapid ventricular rate (above 230 per minute) with extremely short diastole and weak contractions, reducing the cardiac output, as in (*a*) some cases of auricular flutter with rapid ventricular rate, (*b*) paroxysmal tachycardia.

3. Ventricular fibrillation, causing sudden syncopal death due to absence of proper ventricular systole.

4. Aortic valvular disease—more commonly in stenosis than regurgitation; syncope frequently occurs on exertion.

5. Coronary disease with thrombosis or in anginal attacks. (The syncope in many of these cases is due to vasovagal attack).

Adams-Stokes syndrome.—It consists of attacks of syncope with epileptiform convulsion, due to abnormally long pauses between ventricular beats, as in cases of heart-block when the ventricular rate falls below twenty per minute, or when temporary cardiac stand-still occurs. Sudden loss of consciousness without warning, twitching of the face and upper limbs, pallor, and absence of ventricular beats or presence of a very slow ventricular rate are the features of this syndrome.

Cardiac syncope is to be distinguished from:—

(*a*) *Epilepsy.*—by the absence of aura, tongue biting and incontinence of fæces and urine in the former and unchanged pulse in the latter.

(*b*) *Vasovagal syncope.*—by absence of sweating and vomiting and by the presence of heart-block or other organic cardiac defects between attacks. Organic heart disease, however, may be complicated with vasovagal syncope.

(*c*) *Cerebral arteriosclerosis and hypertensive encephalopathy*—by the presence of pallor and slow pulse and absence of headache, paræsthesia, aphasia, paresis etc., or hypertensive pulse.

ANGINAL FAILURE

When the supply of oxygenated blood to the myocardium through the coronary arteries is inadequate, either at rest or in exercise, a characteristic type of prækordial pain or distress and a sense of strangling occurs. This subject will be discussed later on under angina pactoris. (See Chapter VI).

GENERAL VASCULAR FAILURE OR PERIPHERAL FAILURE

A failure of peripheral circulation occurs due to a serious diminution of the volume of blood in effective circulation. This may result in three ways,—

1. Severe internal or external hæmorrhage.

2. Loss of fluid from the body by vomiting, purging etc. or by increased exudation from capillaries into the tissues, as in injuries, burns etc., (*surgical shock*).

3. Stagnation in the widespread capillary network, due to dilatation, either from (*a*) action of toxins, as in infective fevers like pneumonia, diphtheria etc., or from (*b*) nervous influence, as in vasodilatation of vasovagal syncope, or of primary shock.

In the first two circumstances, the anoxæmia from diminished blood-volume also leads to capillary damage and dilatation, and peripheral stagnation. The result is a fall of blood pressure, coldness and cyanosis of the skin specially at the periphery, diminished venous return to the heart, fall of cardiac output, compensatory tachycardia, a low volume and tension of the pulse and cerebral anæmia.

Treatment.—1. Increased venous return to the heart should be helped by (*a*) raising the foot end of the bed, and (*b*) bandaging the limbs.

2. Restoration of blood volume by: (*a*) replacement of lost fluid and salts in cases of dehydration, (*b*) blood transfusion in hæmorrhage, (c) plasma or serum transfusion, specially in injuries, burns etc.

3. *Peripheral circulatory stimulants.*—(*a*) Adrenaline ½ c.c. to 1 c.c. of 1 in 1,000 solution subcutaneously. The effect is short lasting. Repeated injections may be necessary, (*b*) Suprarenal cortical extract improves the peripheral circulation with more lasting effect. Eucortone 10 to 20 c.c. or Percorten 5 mgm. may be given intramuscularly. (*c*) Cardiazol 1 c.c. subcutaneously. (*d*) Coramine 2 c.c. intramuscularly. (*e*) Warmth by hot water bottles, blankets and electric cradles.

CHAPTER III

RHEUMATIC HEART DISEASE

It is estimated that rheumatic infection is responsible for nearly forty per cent of all heart diseases, and for more than ninety per cent of all heart diseases below the age of thirty years. Formerly, the cardiac manifestations were regarded as complications in the course of acute rheumatic fever. It is now recognised that in rheumatic infection the essential features are 1. Acute non-suppurative arthritis with fever, 2. Carditis, 3. Chorea, 4. Subcutaneous nodules. In any particular case, these manifestations may be present in varying degrees and combinations. One or the other may be most prominent either simultaneously or at different periods.

Aetiology.—Rheumatic infection is essentially a disease of childhood and adolescence. Age limits are 2 to 25 years, with maximum incidence in the 7th. and 8th. years. It is said to be more common in female children than in males. In our experience, however, male children are more affected. A familial incidence is noticed in almost fifty per cent of the cases.

Predisposing causes.—1. Unhygienic conditions like ill ventilation, overcrowding, dampness etc.

2. Malnutrition, specially vitamin deficiency.
3. Nervous excitability.
4. Focal sepsis, specially of the upper respiratory tracts.

Exciting causes.—No specific infective agent has yet been definitely found. The lesions have been considered to be due to 1. Streptococcal infection, 2. Bacterial toxins, 3. Filterable virus infection, 4. Allergic reaction to a focal streptococal (β-hæmolyticus) infection or to an infection of a non-specific character.

Whatever may be the cause, a relation to some infection specially of the upper respiratory tract is constantly found. The mode of infection is by droplet inhalation.

Some authorities prefer to call the condition *rheumatic state,* in which there are two principal components. 1. Nervous excitability and 2. Infection. The former is manifested in chorea and the latter in acute inflammatory lesions of the joints or in acute or subacute lesions of the heart. These may be present in a particular case in varying degrees.

Pathology.—The disease affects the connective tissues of the body. Two types of lesions are seen, exudative and proliferative. The former is seen as effusions in joints and serous sacs specially the pericardium. The latter is seen as small submiliary nodules called the *Aschoff's nodes.* These constitute the essential specific lesions of rheumatic disease.

The Aschoff's nodes develop in connection with small vessels and are seen best in the myocardium. They consist of a central area of collagenous necrosis surrounded by a number of large mononuclear cells with large vesicular nucleus. A few multinuclear cells with two or more nuclei resembling the Hodgkin giant cells are also seen. In the periphery, plasma cells and lymphocytes also collect. Varying degrees of fibroblastic reaction are seen and in prolonged cases there is considerable fibrosis.

Morbid changes are chiefly found in the heart and the joints.

Heart.—All the structures may be affected (*carditis*), but the severity may be more in one than the other.

Endocardium.—A valvular endocarditis occurs, affecting in order of frequency the mitral, aortic and very rarely the tricuspid and pulmonary valves. In the acute stage, a row of sessile bead-like vegetations are seen along the line of contact of the cusps during closure. The vegetations consist of conglomerated blood platelets and leucocytes enmeshed in fibrin. These may organise and cause scarring of the valves leading to deformity and shortening of the cusps, shortening and thickening of the chordæ tendinæ and hence incompetence of the valves during closure. Adhesions between cusps and contraction may lead to stenosis. These changes are specially liable to occur in prolonged subacute or relapsing cases. A mild attack, however, leaves no traces or only slight scarring of the valves specially the mitral, which does not interfere with the functional efficiency.

Mitral valves are said to be affected in all cases of rheumatic infection, but permanent changes ranging from slight thickening of the cusps to advanced stenosis may or may not be left.

Myocardium.—In the acute stage, myocarditis with scattered Aschoff's nodes and cloudy swelling of the muscle fibres occurs. Later on slight fibrosis may be left, but myocardial inefficiency may develop at any stage and may not be associated with any marked demonstrable pathological changes.

Pericardium.—Fibrinous or serofibrinous pericarditis may occur. Localised fibrosis or adhesions may be found later in the pericardium as milk spots. Sometimes, extensive adhesions between the two layers of the pericardium and the mediastinal structures, chest-wall, diaphragm and pleura may develop. This mediastino-pericarditis or chronic adhesive pericarditis may lead to overwork and enlargement of the heart. A chronic constrictive pericarditis may also occur, in which there is complete obliteration of the pericardial sac by contracting fibrous tissue with occasional calcification. In such cases, the orifices of the superior or inferior vena cavæ are obstructed and there is general compression on the heart.

Joints.—A serous effusion in the large joints may develop. It never suppurates and leaves no permanent deformity.

Other lesions.—Occasionally, a serous pleural effsion may occur; and a peculiar hæmorrhagic consolidation of the lung has been described as rheumatic pneumonia.

Clinical manifestations.—This may be acute, subacute and chronic.

Acute.—It is usually seen as acute rheumatic fever with abrupt onset of fever with sorethroat, and pain and swelling in one or more joints, such as the knees, ankles, wrists, elbows, shoulders or hip joints, which are usually involved in succession. There is profuse acid sweat. Fever runs an irregular remittent course and leucocytosis is usual.

The cardiac involvement is manifested by carditis. The pulse is rapid, out of proportion to the temperature. The apical impulse moves outwards. A systolic murmur may appear at the apex and the first sound may be muffled. Cardiac irregularities may appear. Signs of congestive failure may develop in severe cases. A pericardial friction rub or signs of pericardial effusion may develop.

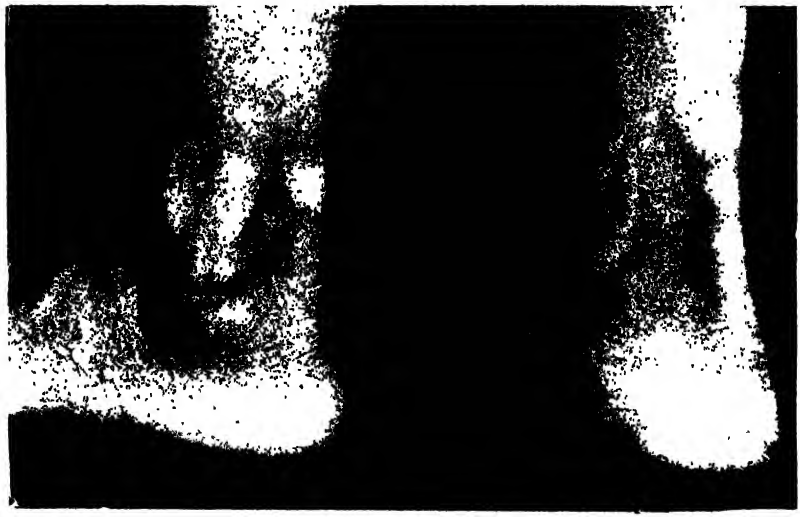


Fig. 9. Rheumatic nodules.

This acute type is seen mostly in adults and the affection is more in the joints than in the heart. The joint manifestations and fever may subside, but the infection may persist as a subacute affection of the heart with progressive and permanent damage to its structures.

Subacute.—This is mostly seen in children. Onset is insidious. Fever and joint pains are slight or even absent. Malaise with or without muscular and joint soreness or 'growing pains' and chronic sorethroat are present. Anæmia and loss of weight are usual but

may not be sufficient to attract immediate attention. The child may not be sufficiently ill to prevent school-going. Rheumatic nodules (see page 13) may be detected on examination. In some children, chorea may occur.

Progressive changes in the heart may proceed for months or years without much outward manifestations of infection until symptoms of cardiac failure appear to call for a detailed examination of the child. This may reveal an enlarged heart with or without a recognisable valvular defect depending on the duration of the disease. A clear history of arthritis is absent in such cases. The

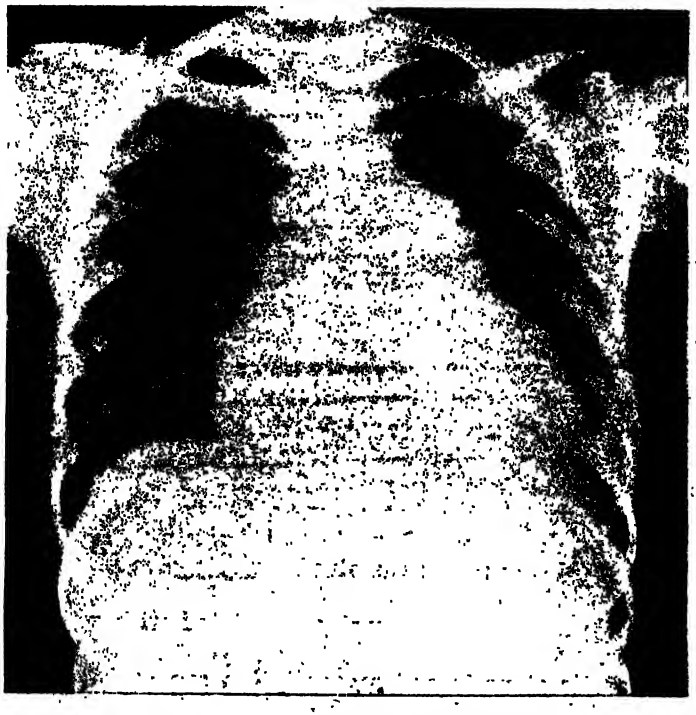


Fig. 10. Shadow of the heart in mitral stenosis as seen under X-rays.

activity of the infection may be assessed by the following signs:—

1. Any elevation of temperature above 99°F.
2. A persistently rapid pulse rate, even during sleep.
3. Persistence of joint pains.
4. Lack of energy and loss of weight.
5. Anæmia and leucocytosis.
6. High sedimentation rate of the red corpuscles (above 12 mm per hour).

Chronic.—This is seen mostly as chronic valvular diseases and chronic pericardial changes with or without cardiac failure, and cardiac irregularities. Symptoms are absent unless cardiac failure or some other complications are present.

Valve defects.—These are commonly mitral stenosis and regurgitation, aortic regurgitation, and aortic stenosis.

Mitral disease is more constant and aortic lesions are usually combined with it. Rarely aortic lesions are found alone.

Mitral stenosis.—It develops as a result of progressive or repeated affection of the mitral valves. It takes on an average two years from the onset, for obvious stenosis to develop. Sometimes the changes in the valve do not progress beyond shortening and thickening. This along with shortening of the chordæ tendinæ causes mitral regurgitation, but there is no stenosis of the mitral orifice.

Signs of mitral stenosis.—1. Presystolic murmur (see page 23) and thrill in the mitral area. 2. Short and sharp first sound. 3. Slapping apical impulse. 4. Accentuation of the pulmonary second sound. 5. A systolic murmur of mitral regurgitation may be associated. Enlargement of the pulmonary conus as seen by X-rays. (Fig. 10).

Aortic regurgitation. *Signs.*—1. Typical aortic diastolic murmur with conduction downwards along the left border of the sternum. (See page 24). 2. Heaving apical impulse with shifting of the apex downwards and outwards. 3. Water-hammer pulse. 4. Exaggerated carotid pulsation. 5. Capillary pulsation. 6. Duroziez's (diastolic) murmur over the femoral artery.

Aortic stenosis. *Signs.*—1. Rough and loud aortic systolic murmur conducted into the neck. 2. Systolic thrill in the aortic area. 3. Left ventricular hypertrophy (Apex beat shifted downwards, heaving impulse and prolonged booming first sound). 4. Typical pulse (slowly rising sustained hypertensive pulse often with anacrotism).

The aortic lesions are generally associated with mitral stenosis and its signs.

When aortic stenosis and regurgitation are both combined, the pulse may not be typical of either condition, but inclines more towards the more prominent lesion.

Pericardial changes.—Sometimes, chronic rheumatic disease is seen as chronic mediastino-pericarditis or rarely constrictive pericarditis. Symptoms are those of cardiac failure or venous obstruction.

Mediastino-pericarditis. Signs.—1. Systolic retraction of the præcordium and the lower intercostal spaces in the lateral or posterior chest wall (*Broadbent's sign*). 2. Fixed apical impulse; not moving with change of position (specially seen under the X-ray screen). 3. Cardiac enlargement and failure. 4. Pulsus paradoxus. 5. Venous congestion.

Chronic constrictive pericarditis (Pick's disease). Signs.—Œdema of the legs. 2. Ascites. 3. Enlarged liver. 4. Engorged neck veins (without pulsation). 5. Dyspnœa may be slight or absent. 6. Heart size is normal and no evidence of valvular disease is present. 7. X-ray shows thickening of the pericardium and sometimes streaks of calcification.

For detailed signs see under chronic pericarditis (Chapter VIII).

Course, prognosis and complications.—Acute attack is rarely fatal. Prognosis depends on the degree of cardiac involvement, which bears no relation to the severity of fever or joint affections. The longer the attack, the more is the cardiac damage. Relapses are very frequent and younger the age, greater is the liability of relapse. With each relapse, more cardiac damage occurs. Even in the absence of more prominent clinical symptoms, the heart may not be free from active disease.

In subacute cases, heart damage may proceed unsuspected. At any stage, the prognosis depends on the degree of cardiac reserve and on the persistence of active infection or the chances of its relapse.

The possibility of permanent cardiac damage developing in rheumatic fever is very great and so also in subacute cases and in chorea.

In chronic rheumatic heart disease, the prognosis depends again on the exercise tolerance, size of the heart and chances of relapse. In all chronic valvular diseases, signs of failure and signs of activity of the infection should be looked for. In the presence of these the outlook is gloomy. In their absence, the chances of relapse should be considered. Younger age and persistence of the predisposing causes render the patient liable to relapses.

In cases with valvular disease alone without any failure or signs of activity of the disease, the prognosis is better in mitral than in aortic disease.

Mitral stenosis may be complicated with auricular fibrillation which may precipitate failure.

Subacute bacterial endocarditis is a fatal complication which makes the prognosis in all valvular diseases to a certain extent uncertain.

Treatment.—No specific drugs are known, which can eradicate rheumatic infection.

Treatment in acute and subacute cases consists in 1. Rest. 2. Hygienic measures. 3. Symptomatic relief.

Rest.—Strict bed-rest is necessary to give rest to the heart. Even in cases without any cardiac manifestations, the patient should be in bed-rest until all signs of activity of infection have disappeared. In cases with obvious signs of cardiac involvement rest should be prolonged sufficiently to enable the heart to recover completely (3 to 6 months after disappearance of all signs of activity).

General hygienic measures.—These should be such measures as are necessary for treating any patient in bed for a long time. These include oral hygiene, attention to bowels, avoiding bedsores etc.

Removal from unhygienic home surroundings, proper ventilation and adequate nourishing diet are also necessary.

Symptomatic relief. Fever and joint pains.—Sodium salicylate acts almost as a specific. It should be given in large doses, 120 to 240 grains in twentyfour hours, at frequent intervals. It should be combined with adequate amounts of alkalies to protect the stomach from irritation. Salicylates have no action on the heart lesions. The affected joints should be kept warm and at rest.

Cardiac failure.—Besides physical and mental rest, attempt should be made to lower the pulse rate with digitalis. As these cases are acute, intravenous digoxin or strophanthin is indicated in presence of œdema. Large doses of glucose by mouth should be given. Oxygen inhalation may be necessary.

During convalescence, the patient should be kept at rest sufficiently long to ensure complete recovery of the heart. The period of rest should extend four to six weeks after all signs of activity of infection have disappeared. In cases, when heart has been obviously affected, the rest period should be increased to three to six months.

Nourishing diet and adequate vitamins should be given and any anæmia present should be corrected by iron preparations.

Septic foci, specially grossly infected tonsils should be eradicated.

In subacute cases, treatment is mainly rest, symptomatic relief and general hygienic measures.

CHAPTER IV

CARDIOVASCULAR SYPHILIS

Cardiovascular lesions of syphilis become manifest on an average twenty years after the primary infection. Only a small number of syphilitics (10%) develop these lesions. Age incidence is above forty years. It may be a decade earlier in some cases in the tropics. Females are rarely affected.

Pathology.—The disease starts in the wall of the ascending aorta at its origin. There is a chronic inflammation with lymphocytic and plasma cell infiltration in the media and adventitia, and endarteritis obliterans of the vasa vasorum. This is followed by gradual degeneration of the elastic tissue and formation of fibrous tissue with consequent thickening and scarring of the aortic wall. Weakness from loss of elasicity, leads to stretching and dilatation. The fibrosis and scarring also extend into the intima, which loses its normal smoothness. Fibrous thickening at the orifices of the branches of the aorta causes narrowing, which is of great importance when the coronary openings are involved.

Starting from aortitis three important results may follow:

1. Extension downwards to the aortic orifice and aortic valves of the heart. The valve cusps are fibrosed and retracted into the wall of the aorta at their attached margins causing separation of the cusps. The destruction of the elastic ring around the aortic orifice leads to dilatation and further separation of the cusps at the commissure. *Aortic regurgitation* is the result. As the cusps never get adherent, stenosis never occurs.

2. Intimal scarring extending into the sinuses of Valsalva causes *narrowing of the coronary ostia.* Angina pectoris, coronary thrombosis and sudden death are the results. The disease, however, does not extend into the coronary arteries.

3. Extension upwards into the ascending aorta, aortic arch or descending aorta, causes dilatation, which may remain localised in one portion or may be diffuse. A *saccular aneurysm* or a *fusiform aneurysm* may thus be formed.

Besides these lesions, a gumma may occasionally form in the heart muscle.

Clinical manifestations.—In the earliest stage of aortitis, symptoms and signs may be absent. Occasionally, retro-manubrial pain or discomfort may be present specially at night.

When aortic dilatation is present, the signs are:

1. Accentuated and ringing aortic second sound. 2. Aortic systolic murmur. 3. Widening of the paramanubrial dullness on percussion. (This may not be present unless the dilatation is well-

marked). 4. X-ray or fluoroscopic evidence of widening of aortic shadow.

In the advanced stages, the manifestations are those of 1. Aortic regurgitation. 2. Coronary insufficiency. 3. Aneurysm of the aorta.

Aortic regurgitation. *Symptoms.*—Except palpitation and throbbing in the head and neck due to forcible cardiac action and high pulse pressure symptoms are generally absent until cardiac failure supervenes. Patients come with cardiac asthma and less commonly with breathlessness on exertion. Some patients complain of anginal pain, and others of giddiness or fainting specially on exertion.

Signs.—Cardiac signs.—(a) Aortic diastolic murmur with characteristic propagation (see page 24). Usually, an aortic systolic murmur is also present due to dilatation of aorta, giving a to-and-fro character.

(*b*) Signs of left ventricular enlargement and hypertrophy.

(*c*) Apical systolic murmur may be present due to relative mitral regurgitation. Occasionally, the Austin Flint murmur of relative mitral stenosis is present.

Peripheral signs.—(*a*) Water-hammer pulse. (*b*) Exaggerated pulsation in the carotids and the peripheral arteries. (*c*) Capillary pulsation. (*d*) Duroziez's murmur and systolic pistol shot sound on the femoral artery.

Coronary insufficiency.—This is usually seen as angina of effort. Aortic regurgitation is generally present in these cases. Coronary thrombosis is rare.

Aneurysm of aorta.—Aneurysms manifest themselves by certain local signs and by pressure effects on the surrounding structures.

Symptoms.—Subjective symptoms are produced by pressure effects on the surrounding structures and due to heart failure from associated aortic regurgitation and coronary insufficiency.

The common presenting symptoms are pain, breathlessness, dry cough, hoarseness of voice, hæmoptysis and dysphagia.

Pain.—This may be due to (*i*) stretching of the aortic wall—a retrosternal oppression, (*ii*) erosion of bone—a constant gnawing pain, and (*iii*) irritation of nerve roots—neuralgic pain.

Breathlessness.—It is due to pressure on the trachea, or from associated heart failure.

Dry cough.—It is due to pressure on the trachea.

Hoarseness of voice.—It is due to paralysis of the recurrent laryngeal nerve, specially of the left-side, in aneurysm of the arch of aorta.

Haemoptysis.—It is due to erosion of the trachea.

Dysphagia.—It is due to pressure on the œsophagus.

Local signs. 1. *Aneurysm of ascending aorta* (aneurysm of signs).

(*a*) Pulsatile swelling, commonly in the second intercostal space on the right side of the sternum. The swelling may be tender with inflammation of the skin over it.

(*b*) Systolic thrill and diastolic shock over the swelling on palpation.

(*c*) Paramanubrial dullness on percussion.

(*d*) Systolic murmur over the swelling.

(*e*) Systolic and diastolic murmurs in the aortic area, due to aortic regurgitation.

(*f*) Accentuated ringing aortic second sound.

Fig. 11. Aneurysm of the ascending aorta as seen under X-rays.

2. *Aneurysm of the arch of the aorta* (aneurysm of symptoms). A pulsatile swelling is generally absent due to deeper position. Pulsation in the suprasternal notch may be seen. Widening of the paramanubrial dullness is present. A downward pull on the trachea with each systole is felt if the cricoid cartilage is held between the fingers from behind (*tracheal tugging*).

3. In aneurysm of the descending aorta, no local signs are present.

Pressure signs.—Effect of pressure on various structures in the mediastinum. (*a*) *Trachea.*—Pressure causes breathlessness, stridor, hæmoptysis (from erosion).

Fig. 12. Aneurysm of the aortic arch and descending aorta as seen under X-rays.

(*b*) *Bronchus.*—Pressure is more commonly on the left bronchus by aneurysm of the aortic arch. Deficient breath sounds at the left base and sometimes signs of collapse and bronchiectasis.

(*c*) *Œsophagus.*—Dysphagia, rarely severe enough to cause difficulty in swallowing even liquids.

(*d*) *Left recurrent laryngeal nerve.*—Paralysis of the left vocal cord and hoarseness of voice.

(*e*) *Cervical sympathetic.*—In early stage, pressure causes irritation, producing dilatation of pupil, retraction of upper eye-lid and exophthalmos on the same side; later there is paralysis producing contraction of pupil, ptosis and enophthalmos (*Horner's syndrome*).

(*f*) *Thoracic nerve roots.*—Pain along intercostal nerves.

(*g*) *Superior vena cava.*—Puffiness of the face, engorged neck-veins and dilated veins on the chestwall.

(*h*) *Bones.*—Sternum may be eroded. In aneurysm of the descending aorta, erosion of the vertebral column with local pain, and in extreme cases, pressure on the spinal cord causing paraplegia.

(*i*) *Large arteries arising from the aortic arch.*—Inequality of the pulse on either wrist, being small on the affected side, and inequality of pupils, being dilated on the affected side.

X-ray examination.—A shadow of the same density and continuous with the shadow of the aorta is seen. Its outline is smooth, rounded or conical and shows pulsation under the screen. (See figures 11 and 12).

Differential diagnosis.—Syphilitic aortic regurgitation must be distinguished from regurgitation of rheumatic and degenerative origin.

TABLE IV

	Syphilitic.	Rheumatic.	Atheromatous
1. Age.	Above 40 years.	Below 35 years.	Above 50 years.
2. History of			
(*a*) Syphilis.	Present.	Nil.	Nil.
(*b*) Rheumatic infection.	Nil.	Present.	Nil.
3. Mitral Stenosis.	Absent.	Usually present.	Absent.
4. Aortic Stenosis.	Absent.	May be present.	May be present. (with evidence of calcification of the aortic valves).
5. Degree of regurgitation.	Free.	Moderate.	Slight.
6. Aneurysm of aorta.	May be present.	Absent.	Absent.
7. W. R. of blood.	Positive.	Negative.	Negative.

Aneurysm of the aorta must be distinguished from mediastinal growths or enlarged glands. The diagnosis depends on the age of the patient, history of syphilis, absence of any wasting, and presence of cardiovascular signs of associated aortic regurgitation. When local

signs, like pulsatile swelling are present diagnosis is confirmed. X-ray examination also helps in diagnosis.

Complications. *Aortic regurgitation.*— Complications are (*a*) heart failure, (*b*) sudden death, (*c*) rupture of aortic valves, (*d*) sub-acute bacterial endocarditis.

Aneurysm of the aorta.—Complications are (*a*) rupture, (*b*) thrombus formation inside the sac and embolism.

Coronary thrombosis is a rare complication of syphilitic aortitis, and cardiovascular syphilis may be associated with neurosyphilis.

Prognosis.—When obvious manifestations are present, outlook is gloomy. Angina and aneurysm have the worst prognosis, although some cases are seen to survive a pretty long time after the manifestations of aneurysm. In individual cases, the prognosis depends on the severity of symptoms, degree of cardiac failure and response to treatment. Average duration of life after diagnosis is three to four years.

Treatment. *General treatment.*—Rest, both physical and mental should be enforced in all cases. All sorts of physical exertion or mental excitement should be avoided even in the absence of cardiac failure. Moderation in food and drink is necessary.

Specific treatment.—Presence of gross cardiac failure, angina pectoris or advanced aneurysm contraindicates specific treatment. If cardiac failure responds sufficiently well to proper treatment, then very cautious specific treatment should be given.

In early cases, as in aortitis or mild cases of aortic regurgitation or aneurysm, antisyphilitic treatment may be given cautiously to arrest the progress of the disease.

Arsenical preparations should not be given first owing to a danger of sudden increase of the inflammatory reaction around the coronaries causing sudden death (*Jarisch-Herxheimer reaction*). A preliminary course of potassium iodide gr. 15 to 30 t.d.s. by mouth and bismuth injections weekly for twelve weeks (dose 0·1 gm. for four injections and 0·2 gm. for eight injections) should be given. This is followed by a course of sulpharsphenamine injections starting from 0·1 gm. and increasing to a maximum of 0·4 gm. by weekly intramuscular injections for twelve weeks. The course of bismuth and arsenic injections should be repeated after three months' interval, and then at six months' interval for the rest of the patient's life for a permanent arrest.

The usual precautions against toxic complications of bismuth and arsenic should be taken. The mouth should be kept clean and regular examination of urine should be done for the presence of albumin and casts.

CHAPTER V

HYPERTENSION AND HYPERTENSIVE CARDIOVASCULAR DISEASE

An increase in both the systolic and diastolic pressure above the normal maximum limits (systolic 150 mm. and diastolic 90 mm.) is hypertension. Increase of systolic pressure alone is not true hypertension and may occur temporarily in emotional excitement, physical exertion or after meals, thyrotoxicosis, complete heart-block and severe arteriosclerosis (due to loss of elasticity of the vessels).

Causes of true hypertension are—1. *Essential hypertension* or constitutional hypertension (*Hyperpiesia of Allbutt*).

2. *Kidney diseases.*—(*a*) Acute, subacute and chronic diffuse glomerulonephritis. (*b*) Polycystic kidneys. (*c*) Chronic urinary obstruction. (*d*) Bilateral pyelonephritis or hydronephrosis.

3. *Toxic causes.*—(*a*) Eclampsia and pre-eclamptic toxæmia. (*b*) Lead poisoning.

4. *Endocrine disturbances.*—(*a*) Pituitary basophil adenoma (*Cushing's syndrome*). (b) Suprarenal tumours—(*i*) pheochromocytoma (Paroxysmal hypertension), (*ii*) Cortical adenoma. (*c*) Ovarian dysfunction (climacteric hypertension).

5. *Central nervous causes.*—(*a*) Increased intracranial tension. (*b*) Midbrain and brain stem lesions, as in bulbar poliomyelitis.

6. *Coarctation of the aorta.*—(Hypertension only in the upper extremities).

Except essential hypertension and nephritis, hypertension due to other causes is much less common.

Prolonged hypertension, from whatever cause it may be, has the same injurious effects on the heart and the blood vessels. In speaking of hypertensive cardiovascular disease, essential hypertension is generally referred to. This term was orginally used to distinguish cases of hypertension not of nephritic origin.

ESSENTIAL HYPERTENSION

Aetiology. *Age.*—Persons above forty years are commonly affected.

Sex.—Both sexes are equally liable.

Race and nationality.—No restriction is seen. It is said to be less common amongst the Chinese and African Negroes. Although it has been said to be less common in the tropics, it is by no means so in our experience.

Occupation.—Although no occupation is exempt, it is more commonly found in the higher strata of society and in persons engaged in intellectual work rather than in physical exertion.

Heredity.—Hereditary and familial incidence is very common. It is believed that a particular inherited constitutional peculiarity acts as the main predisposing factor.

Constitutional and physical habitus.—Most hypertensives have the sthenic type of constitutional habitus, and hypertension is more common in obese than in thin people.

Diabetes and gout.—The association of hypertension with either or both of these in the same person or in different members of the same family is quite common. It is possible that the same inherited constitutional type predisposes to obesity, gout, diabetes and hypertension.

Certain other factors, which from time to time have been regarded as the cause of hypertension, act as aggravating or precipitating agents in the presence of the constitutional predisposition. These are (*a*) emotional disturbances, worries and anxieties, overwork and insufficient sleep, (*b*) habitual overeating, and (*c*) excessive smoking.

Pathogenesis.—There is evidence to show that the raised arterial tension in essential hypertension is due to arteriolar constriction. The constriction, however, is not sufficiently severe to reduce the amount of blood flow through the skin or the organs, except perhaps through the kidneys. On the other hand, the amount of blood flow through various organs and tissues is maintained within normal limits. The constriction may be organic, nervous or chemical in origin. The evidences so far available suggest that it is neither organic, nor nervous, but is due to chemical factors. None of the known chemical pressor substances has an effect on the skin and renal blood flow similar to that seen in essential hypertension. A substance present in extracts from kidneys (specially ischæmic kidneys), however, produces an effect on renal blood flow identical to that seen in essential hypertension. This substance is called renin. Its role in essential hypertension is however still uncertain.

Pathology.—The underlying pathology of hypertension, like its cause is unknown. Probably, it begins primarily as a functional spasm of the arterioles and leads to pathological changes which are but natural consequences of prolonged hypertension of any origin.

The main changes are seen in the heart and the bloodvessels of the various organs such as the kidneys, brain and the heart.

Heart.—The first effect on the heart is a hypertrophy of the left ventricular musculature. The individual muscle fibres increase in size, but there is no increase in number. The left ventricular cavity is not enlarged, so that compared with the thickness of the wall the cavity appears small (so-called concentric hypertrophy). There is increase in the weight of the heart (normal being 200 to 300 gms.) and in the thickness of the left ventricular wall (normal being 12 to 15 mm.).

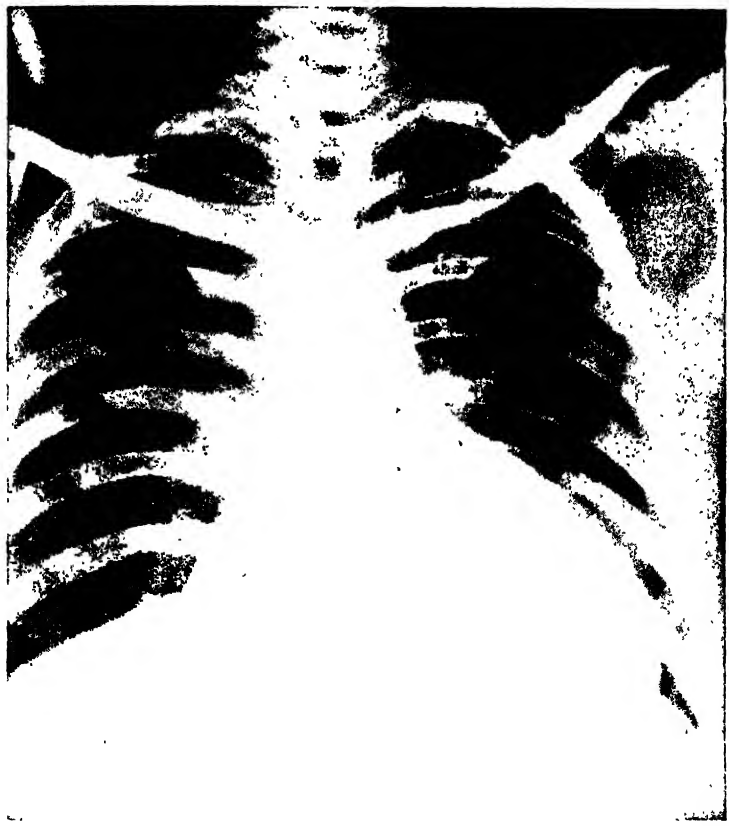


Fig. 13. Left ventricular enlargement as seen under X-rays.

Later, when myocardial efficiency fails due to lack of adequate blood supply as a result of concomitant coronary changes or pro-

longed strain, there is enlargement of the left ventricular cavity. The mitral ring also enlarges with left ventricular dilatation causing relative mitral regurgitation. The dilatation is shown by increase in size and capacity of the left ventricle.

The left ventricular failure and relative mitral regurgitation lead to pulmonary congestion and increased work for the right ventricle. Ultimately, right ventricle also enlarges and failure with congestion in the systemic veins develops. The heart shows an enlargement in all the diameters.

Blood vessels.—The following types of changes are seen in blood vessels.

Atheroma.—This is seen in the large elastic arteries like the aorta and its large branches, and in the small and medium sized arteries of the organs, specially the coronary and the cerebral arteries. The heaped up fatty and cholesterol deposit in the subendothelial layer of the intima, which in large arteries, as the aorta, is not of much significance, produces serious narrowing of the lumen in the coronaries and the cerebral vessels. Ischæmic degeneration in the myocardium or softening in the cerebral substance may result. More important still is the development of thrombosis in these vessels, which produces dramatic clinical manifestations.

Associated degenerative changes in the media with consequent loss of elasticity, lead to stretching and elongation, as in the aorta and render the cerebral arteries liable to rupture, specially when there is loss of support from surrounding ischæmic softening or when there is a sudden rise of blood pressure.

Although atheroma is more common and severe in cases of hypertension, it can occur without it.

Diffuse hyperplastic sclerosis.—This change is much more closely related to hypertension than atheroma, as it is seldom encountered in the absence of hypertension and is constantly found in hypertension of some duration. It affects mainly the arterioles and is therefore called *arteriolosclerosis.* All the arterioles are not equally affected. Those of the kidneys are most constantly affected and those of the spleen, pancreas and brain to much less an extent.

There is hyperplasia of the internal elastic lamina, which becomes reduplicated. Subendothelial proliferation and hyaline deposition cause gradual obliteration of the lumen. Some fibrosis in the media occurs following a hyperplasia of the muscular tissue.

Kidneys.—In the kidneys, the interlobular and the afferent glomerular arterioles are affected. Their obliteration leads to atrophy and hyaline degeneration of the glomerulus, atrophy of the corresponding tubules and subsequent replacement of the degenerated elements

by fibrous tissue. This in turn contracts and produces depressions on the surface, giving rise to the appearance of granular contracted kidneys, also known as arteriolosclerotic kidney or primary contracted kidney (chronic interstitial nephritis of older writers).

In one variety of hypertension, the vascular changes are of a more severe and acute character. These cases are described as *malignant hypertension* in contradistinction to the more slowly progressive type of *benign hypertension,* which produces the changes described above. In malignant hypertension, the arterioles show a necrotising arteriolitis with deposit of a necrotic granular mass in the media and intima, reducing or occluding the lumen. These changes are mostly seen in the kidneys, leading to rapid renal failure. The kidneys may show varying degrees of granular change depending on the duration of hypertension and degree of arteriolosclerosis; but they are generally uncontracted and hæmorrhagic spots on the surface may produce a 'fleabitten' appearance.

Symptoms and signs.—From the pathology, it will be evident that the clinical manifestations of hypertension will be either cardiac failure, coronary insufficiency, cerebral vascular lesions or renal failure. As the pathological effects are not uniform in all cases, the signs and symptoms will also be diverse. Often, a case of hypertension has no symptoms in the early stages; the blood pressure is detected in the course of routine examination, and symptoms appear after the patient has been apprised of it. Symptoms may be classified under the following headings. Any one of these groups may mark the onset.

Nervous symptoms.—Headache, giddiness, weakness, insomnia, tinnitus, lack of concentration, mental irritability, failure of memory or psychological disturbances may occur due to cerebral arterial changes. Transient cerebral symptoms as coma, paresis, aphasia, amaurosis, or epileptiform fits due to cerebral vascular spasm may occur (hypertensive encephalopathy). Not uncommonly the first manifestation is apoplexy.

Cardiac symptoms.—Palpitation and breathlessness on exertion or cardiac asthma may be the early symptom. Rarely angina pectoris may occur first.

Visual symptoms.—Loss of vision, partial due to retinal changes, or total from retinal hæmorrhage may occur and may be the first symptom in some cases.

Peripheral vascular disturbances.—Tingling or numbness of extremities, coldness, burning sensation or muscular cramps may occur.

Urinary symptoms.—Polyuria specially at night is an early symptom of gradually contracting kidneys. Symptoms of uræmia

(see under uræmia. Part III.) may occur specially in malignant hypertension.

Haemorrhagic symptoms.—Epistaxis is frequently met with as a symptom. Hæmaturia, hæmoptysis, menorrhagia etc., are rarely seen.

Signs.—The most important sign is the high tension pulse, and the high blood pressure is obtained by the sphygmomanometer. The height of the pressure is very variable, the systolic being anything between 160 to 300 mm. and diastolic 100 to 140 mm. The heart shows signs of isolated left ventricular hypertrophy (apex-beat shifted downwards and heaving in character, with loud booming first sound) and failure or congestive failure of both ventricles depending on the stage of the disease. (See page 37).

Electrocardiographic changes of myocardial damage due to coronary narrowing are commonly present.

The peripheral arteries like the brachial, radial, temporal etc., may feel cordlike, hard and tortuous. The changes are however more constantly seen in the retina. Evidence of sclerosis of retinal arteries with or without exudate in the retina may be found. Hypertensive neuro-retinopathy especially with papillœdema is commonly seen in malignant hypertension. (See page 32).

The urinary changes depend on the degree of renal damage. Usually, slight albuminuria and some hyaline casts are present. In severe cases, hyaline and granular casts and red blood cells may be present. When marked nephrosclerosis has developed diminishing the concentrating power of the kidneys, a compensatory polyuria develops. Urine is of low specific gravity. Due to improved circulation at rest and during sleep, urinary secretion increases at night. In cases with renal failure, blood urea and non-protein nitrogen are increased.

Complications.—The chief complications have already been mentioned. They are so constant that they find place in the symptomatology of hypertension. These are cardiac failure, cerebral vascular lesions like cerebral hæmorrhage or thrombosis, coronary thrombosis and uræmia. Besides, hypertension may be associated with diabetes, gout and emphysema of lungs.

Diagnosis.—Recognition of hypertension is easy enough provided the sphygmomanometer is used. The important point is to keep it in mind or to use the sphygmomanometer as a routine in the examination of elderly patients. When hypertension has been found, its cause is to be determined. Most cases of hypertension due to a definite cause like endocrine disturbance etc., are easily recognised by the associated characteristic signs. Considerable difficulty may arise however in the diagnosis of essential hypertension from

chronic nephritis. The chief differential points are shown in the following table.

TABLE V

	Essential hypertension.	Chronic glomerulo nephritis.
1. Age.	Usually above 40 years, rarely below 30 years.	Usually below 30 years.
2. History.	No previous history suggesting acute or subacute nephritis.	History suggestive of acute or sub-acute nephritis present.
3. Family history.	History of high blood pressure, apoplexy, cardiac asthma etc. in other members of the family.	No family history.
4. Blood pressure.	More labile.	More constant.
5. Urinary changes.	Changes are slight and not constant in all cases.	More constant and marked changes.
6. Blood chemistry.	Slight increase of urea, N.P.N. etc. or none.	Marked rise of urea, N.P.N. etc.
7. Uraemia.	Less common termination.	More common termination.

The diffculty arises, however, in those cases of chronic nephritis when the early stages have been latent so that no definite history is present. Such cases are difficult to distinguish from malignant hypertension which occur at an earlier age and show marked urinary and blood changes.

Difficulty also arises in those cases, where with the onset of cardiac failure the blood pressure falls. A hypertrophied heart without any evidence of valvular defect is found. Evidence of retinal or renal arteriosclerosis may help in those cases.

Course and Prognosis.—The average duration of life in cases of hypertension is difficult to determine as the onset of the disease is not marked with symptoms. These appear only when sufficient cardiac or vascular damage has occurred. Cases vary greatly in their rate of progress of changes in these structures, and are described as *benign* or *malignant* accordingly. In benign hypertension, changes occur slowly and the patient lives for a considerable time. Vascular changes specially in the kidneys are slow and cardiac failure or cerebral hæmorrhage are the usual modes of death. In malignant

hypertension, vascular and kidney changes progress rapidly with the development of rapid renal failure and neuro-retinopathy.

Prognosis depends on many factors such as:—

Age.—At lower ages, the course is more rapid and tends to be malignant.

Sex.—In females, hypertension runs a milder course.

Height of blood pressure.—Constant higher pressure is more harmful.

Mental attitude.—Too much anxiety or worry about the blood pressure is harmful to the patient.

Infections.—Secondary infection of the respiratory tract or elsewhere may precipitate heart failure.

Anaemia.—This also increases work of the heart and may lead to heart failure.

In any given case, prognosis depends on the condition of the heart, coronary arteries, retina, cerebral arteries and the kidneys.

Heart.—Size of the heart is a rough indication of myocardial condition. In the presence of cardiac dilatation and signs of congestive failure, life is limited. Worst signs are galloprhythm, angina pectoris or electrocardiographic evidence of coronary insufficiency.

Retina.—Hypertensive neuro-retinopathy is found mostly in cases of malignant hypertension and therefore is of bad prognosis. Retinal arteriosclerosis is a better guide to the incidence of changes in cerebral or renal vessels.

Cerebral arteries.—Apart from symptoms of cerebral arteriosclerosis, the condition of the cerebral arteries is difficult to ascertain and therefore lends considerable uncertainty about the prognosis in every case.

Kidneys.—In presence of signs of renal failure expectation of life is very poor.

Causes of death are cardiac failure, coronary thrombosis, cerebral vascular lesion, uræmia and secondary infections.

Treatment.—For treatment, cases of hypertension are divided into two groups—those without any symptoms and those with symptoms.

Cases without symptoms.—It is problematical whether such patients should be apprised of their high blood pressure, because frequently this diagnosis conveys to the patient the constant fear of sudden death by apoplexy or heart failure. The mental upset thus created causes more harm. On the other hand our methods of lowering a raised pressure are far from efficient. All that we can do is to reduce or remove such factors as are known to aggravate or play some ætiological role in hypertension; thereby the progress of changes in the heart and blood vessels can be slowed. The patient should be advised as regards the mode of life he should live, which is a life of moderation avoiding all excesses in diet, exertion, both mental and physical, smoking and drinking, etc. A little explana-

tion and reassurance will do much to allay the anxiety and apprehensions. Vigorous treatment and rigorous restrictions are unnecessary.

General regime. Rest.—The ideal condition is a retired life free from all mental and physical exertion. If this is not possible, avoidance of all strenuous work, limitation of period of work and frequent holidays are necessary.

Exercise.—Mild exercise in the open air in the form of walking is beneficial. Strenuous games are to be avoided.

Diet.—It is not necessary to restrict any particular type of food like proteins or salt. General reduction in quantity so as to avoid gain in weight or in the case of obese persons a reducing diet will be sufficient.

Alcohol and tobacco.—These are better avoided. If total abstinence is too irksome, a moderation in quantity should be made.

Constipation.—A regular action of the bowels should be encouraged in constipated persons by a correction of the diet, so as to include a good quantity of green vegetables, and enforcing a regular visit to the lavatory everyday. Laxatives if necessary, should be prescribed.

Cases with symptoms.—The treatment in such cases besides the general regime, consists in (*a*) measures to lower blood pressure and (*b*) symptomatic treatment.

Measures to lower blood pressure. (*a*) *Physical and mental rest.*—This is the best available method. If mental quietude cannot be obtained otherwise, sedatives should be given. Bromides and chloral hydras may be prescribed in repeated doses, or barbiturates, in the form of phenobarbitone (luminal) 1½ to 3 grains twice a day may be given. This may be combined with codeine phosphate gr. ½ in cases of great nervousness.

(*b*) *Purgation.*—Saline purgatives can lower temporarily blood pressure by repeated fluid evacuations. Saturated solution of magnesium sulphate oz. i every morning should be prescribed in the presence of urgent symptoms due to high blood pressure.

(*c*) *Drugs.*—A large number of drugs have been used from time to time for lowering blood pressure, but only a few have stood the test of time.

The following drugs are extensively used. Some of them have strong hypotensive effect, but this is too temporary to be sufficiently useful.

(*i*) *Nitrites and nitrates.*—The vasodilatation produced by these drugs effectively lowers blood pressure, but the action is temporary. Thus, sodium nitrite in 1 gr. dose by mouth acts in 5 to 10 minutes, but the action lasts only 1 to 2 hours. Amyl nitrite and nitroglycerine are still more transitory in action, but their quick action and coronary dilatation effect are utillised in the relief of angina pectoris attacks. Erythrol tetranitrate in ½ to 1 grain

dose, however, has a longer action for about 5 to 7 hours.

These drugs may cause uncomfortable symptoms like throbbing in the head, palpitation and headache.

(*ii*) *Purine derivatives.*—These drugs also have a vasodilator action especially on the coronary and renal vessels. They are diuretics but their hypotensive effect is not marked. They are however useful in hypertension patients, because they improve the coronary blood flow and thus improve cardiac nourishment. The most effective of these drugs is euphyllin (theophyllin-ethylene-diamine or aminophyllin) used in 1½ to 3 gr. doses.

(*iii*) *Rauwolfia serpentina* (*Sarpagandha*—Sanskrit).—The liquid extract has a strong hypotensive action in doses of 5 to 15 minims t.d.s. The mode of action is by central sedation. The effect is also temporary and does not seem to be constant in all cases.

(*d*) *Venesection.*—Removal of 4 to 6 ounces of blood from a vein is very effective in relieving certain urgent symptoms due to hypertension like cardiac athma and encephalopathy. But the actual lowering of blood pressure is not always appreciable.

Symptomatic treatment.—Symptoms which call for immediate relief are cardiac asthma and hypertensive encephalopathy, congestive cardiac failure, angina pectoris or coronary thrombosis and uræmia.

In hypertensive encephalopathy, the patient should be put to absolute bedrest. 8 ounces of 25 per cent solution of magnesium sulphate should be introduced per rectum as retention enema. If this, with proper sedatives, does not relieve the symptoms, venesection and removal of 4 to 8 ounces of blood should be done. Lumbar puncture with gradual relieving of the intracranial pressure is also useful.

Treatment for other symptoms will be found in the respective sections.

CHAPTER VI

CORONARY INSUFFICIENCY AND ANGINAL SYNDROME

The heart receives its blood supply from the two coronary arteries, right and left, which arise from the aorta at its origin. When the blood supply is inadequate, either at rest or on exertion, the coronary circulation is said to be insufficient. If the resulting ischæmia is of sudden development as when a vessel is suddenly thrombosed or when a narrowed atheromatous vessel is confronted with a sudden demand of increased oxygen supply to the myocardium due to exertion, a characteristic type of præcordial oppression with a sense of strangling occurs, which is described as *angina* (strangling) *pectoris* (chest). Coronary thrombosis as a cause of anginal pain has been recognised more recently and now constitutes a separate clinical entity in the anginal syndrome. The other cases when angina occurs temporarily on exertion are described as *angina pectoris of effort.*

When the coronary insufficiency occurs more gradually due to slow obliteration of the vessels, ischæmic degeneration of the myocardium with subsequent fibrosis occurs insidiously without angina.

Aetiology.—Both effort angina and coronary thrombosis are common in persons above 40 years of age, although not altogether unknown in younger persons. Males are predominantly affected. Stress and strain of modern life, mental worries and anxieties, sedentary habits, excess of tobacco and alcohol, septic foci etc., predispose to anginal syndrome by favouring atheromatous changes in the coronaries. Hypertension, diabetes, obesity and gout are important associates of which hypertension is the most frequent. Of the organic heart diseases, syphilis sometimes produces anginal symptoms due to narrowing of the coronary orifices in the wall of the aorta. Rheumatic heart disease is only rarely associated with angina.

Cause of pain.—The pain is due to myocardial ischæmia. When a vessel is completely blocked, absolute ischæmia and persistent pain occur, as in coronary thrombosis. When, however, a vessel is narrowed by disease or spasm, and the blood supply, although adequate during rest, is incapable of being increased when myocardial activity is increased as on exertion, a relative ischæmia occurs and pain is felt on exertion as in angina of effort. It is a referred pain in which the afferent impulses are carried by the sympathetic through the cervical and upper five thoracic ganglia into the spinal cord.

ANGINA PECTORIS OF HEBERDEN
(EFFORT ANGINA)

This is a sensation of substernal pain and oppression frequently radiating to the left arm and associated with a sense of suffocation lasting for a few minutes, brought on by exertion and relieved by rest.

Causes.—As mentioned, the immediate cause of the attack is myocardial ischæmia. This may be due to:

1. Coronary atheroma. 2. Narrowing at the mouth of the coronary arteries by syphilitic aortitis or atheroma of the aorta. 3. Severe anæmia. 4. Extreme rapid ventricular rate reducing the diastolic period, (period of coronary flow). 5. Spasm of the coronary arteries. 6. Aortic regurgitation and stenosis.

Symptoms and signs.—The characteristic features of the pain have been described before (see page 9). During an attack, the patient is pale and extremely scared with a sense of impending death. He is afraid to move. Most patients are subjects of hypertension and the usual signs are high tension pulse, hypertrophied left ventricle and accentuated aortic second sound. In some cases, signs of cardioaortic syphilis or those of aortic stenosis or regurgitation are present. In other cases, there may be no signs of organic heart disease.

Attacks differ considerably in severity and duration. The intensity of the pain depends more on the nervous excitability of the patient than on the severity of myocardial ischæmia. Attacks may occur regularly whenever a certain amount of exertion is undertaken or there may be attacks at long intervals.

Course and prognosis.—Most cases of angina (about two-thirds) develop coronary thrombosis which may be terminal. Others gradually pass on to myocardial inefficiency and congestive cardiac failure unless they die of cerebral vascular complications of associated hypertension. The severity of the case is judged by the amount of exertion which excites an attack. The greater the coronary narrowing, easier are the attacks produced. Size of the heart and electrocardiographic evidence of the myocardial damage as well as the underlying cause are important in the assessment of prognosis.

CORONARY THROMBOSIS

Causes.—A narrowing of the lumen of the coronary arteries or their branches sufficient to reduce the rate of blood flow beyond the obstruction, favours thrombosis. Such obstruction or narrowing is most commonly due to atheroma of the coronaries or diffuse hyperplastic sclerosis from hypertension. Rarely, atheroma of the aorta or syphilitic aortitis obstructing the coronary orifices may lead to thrombosis. Thromboangiitis obliterans or embolic obstruction of the coronaries due to fragments of cardiac vegetations or torn cardiac valves may rarely be responsible.

In a narrowed atheromatous artery, an atheromatous ulcer or a sudden increase of the atheromatous patch may precipitate thrombosis. A sluggish blood flow, as during sleep, also helps in the development of thrombosis.

Pathology.—Common sites of thrombosis are,—(*a*) anterior descending branch of the left coronary artery, (*b*) circumflex branch of the left coronary, and (*c*) right coronary branches.

The result of thrombosis is sudden loss of blood supply to the portion of the myocardium supplied by the affected vessel. If this is large and a wide portion of the myocardium is involved, the patient dies immediately from ventricular fibrillation. If the vessel is small and the patient survives, an infarct forms in the myocardium. When the anterior descending branch of left coronary is affected, the infarct is situated at the apex extending into the interventricular septum. When the circumflex branch of left coronary or branches of the right coronary are affected, the infarct forms at the base of the heart involving mainly the posterior wall of the left ventricle. The infarct is surrounded by a zone of leucocytic infiltration and if it extends into the pericardial surface, a patch of fibrinous pericarditis develops over it. If it extends into the endocardial surface, a thrombus is deposited over it inside the ventricles, portions of which may be dislodged and cause embolic symptoms. The infarct gradually softens and is removed by phagocytes, and in favourable cases, a scar forms in the wall of the ventricle. Sometimes, the soft infarct may give way under the intraventricular pressure causing sudden death, or the scar may gradually stretch forming a cardiac aneurysm. More often the functional efficiency of the myocardium is impaired due to this death and degeneration of a portion leading to congestive cardiac failure.

Thrombosis of small branches leading to small areas of necrosis and fibrosis may repeatedly occur in the same patient and evidences of this may be seen in the heart on post mortem examination.

Symptoms and signs.—Symptoms depend on the size of the affected vessel. If the main coronary arteries or their large branches are obstructed, there is sudden death of the patient, even before he can feel or speak of the pain. Most of the dramatic instantaneous deaths in elderly persons, especially those with hypertension, are due to this cause.

When a smaller vessel is affected, the patient feels sudden pain in the præcordium exactly of the same character as in angina pectoris. The pain is, however, persistent and is not relieved by rest or amyl nitrite inhalation (see page 9).

Site of the pain may be in the epigastrium, when it is mistaken for acute abdominal conditions.

Along with the pain, there is also acute circulatory failure both peripheral and central. Coldness of extremities, cyanosis, clammy sweats and feeble or imperceptible pulse and a low blood pressure are

present Acute cardiac dilatation with rapid development of pulmonary œdema and venous congestion, dyspnœa etc., may occur. Changes of the first sound in the heart such as weakness and loss of distinctness, tic-tac rhythm and gallop rhythm may be present. Extrasystoles, auricular fibrillation and sometimes heart-block or paroxysmal tachycardia may occur.

If the patient survives the immediate shock and cardiac failure, certain late signs develop, such as a mild degree of fever and leucocytosis due to inflammation around the infarct. A pericardial rub may appear towards the end of the first week and embolism in different sites may occur from the intraventricular thrombus.

Characteristic changes are seen in the electrocardiograph within a few hours of the formation of the infarct. These are of great importance in the diagnosis of coronary thrombosis and in determining the site of the infarct. X-ray shows diminished movement of the left border just above the apex of the heart in apical infarcts.

Relation with angina pectoris.—Coronary thrombosis occurs in a large number of cases of angina pectoris and may be terminal. Less commonly, coronary thrombosis occurs first and is followed by angina pectoris. Rarely, coronary thrombosis ends the anginal attack by destroying completely the ischæmic portion of the myocardium.

Course and prognosis.—Cases vary greatly in their severity. Extensive use of the electrocardiograph in the investigation of hearts of middle-aged people has shown that evidences of old coronary thrombosis are quite common and milder cases frequently occur in which typical signs and symptoms do not occur. Severe cases, however, have a high mortality. Most cases die in the early acute stage from shock, ventricular fibrillation or acute cardiac failure. Of those who survive the early stage, congestive failure develops in many; others may live with severe restriction of exercise tolerance. In mild cases sufficient recovery with return to ordinary occupation in life may occur in a few, but relapses are very frequent. Severity is shown by the duration and severity of pain, degree of shock and cardiac dilatation, pulmonary œdema, marked fall of blood pressure, gallop rhythm and ventricular paroxysmal tachycardia.

Diagnosis.—The main differential points for the distinction of pain due to angina pectoris, coronary thrombosis and neurocirculatory asthenia have been mentioned already (see page 9). Præcordial pain due to other causes such as acute pericarditis, aortic aneurysm syphilitic aortitis and dissecting aneurysm of the aorta can be readily distinguished by the presence of their characteristic signs and by the absence of electrocardiographic evidence of coronary thrombosis. Aortitis and aortic aneurysm may however be associated with true angina pectoris.

Coronary thrombosis may have to be diagnosed from pulmonary embolism. In the latter, there will be a history of recent operation

or injury or a thrombophlebitis. Dyspnœa is more intense and hæmoptysis is common. If the patient survives, the signs of pulmonary infarction will develop. In coronary thrombosis, the electrocardiograph shows characteristic findings.

When the pain in coronary thrombosis occurs low down in the epigastrium, acute abdominal conditions like perforated gastric ulcer or biliary colic and in mild cases acute indigestion may be simulated. In coronary thrombosis, patients are elderly and a history of angina is present, whereas in others a history of ulcer pains or previous attacks of colic or dyspepsia is present. Dyspnœa, cyanosis and signs of cardiac dilatation, arrhythmias, gallop rhythm or other signs of cardiac involvement in coronary thrombosis will contrast with abdominal rigidity, free fluid in the peritoneal cavity and obliteration of liver dullness in gastric perforation. In biliary colic, paroxysmal nature of the pain, prominence of vomiting, absence of cardiac signs or dyspnœa and cyanosis will help.

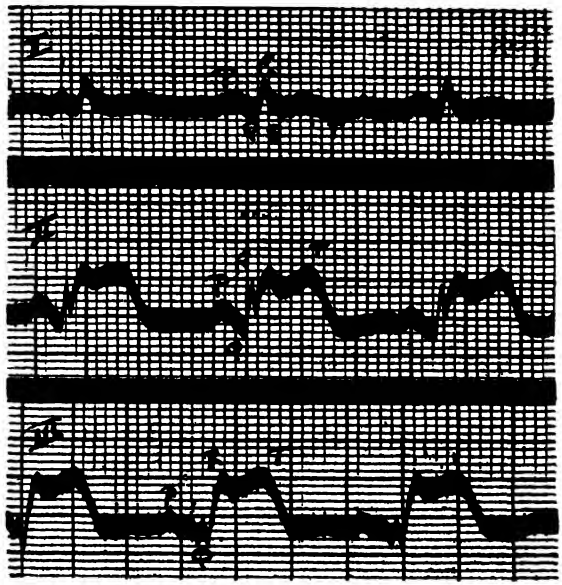


Fig. 14. Electrocardiogram showing coronary thrombosis with basal infarction.

In anomalous cases, when the pain is very mild or is not characteristic, development of late signs like pericarditis, fever, leucocytosis and embolism are of help.

Electrocardiographic findings.—Within a few hours of thrombosis, changes occur in the S-T interval and in the T waves specially

in Leads I and III. In apical infarcts, the S-T segment is elevated above the isoelectric line and T wave is inverted in Lead I and the opposite effects are produced in Lead III. These changes are reversed in Leads I and III in basal or posterior infarcts. (See Fig. 14). Later on, the S-T segment tends to reach the isoelectric line but the T inversion persists for a long time. Sometimes no definite abnormality is seen in the electrocardiogram in the three standard leads. In such cases the chest leads may show abnormalities such as deep inversion of T and elevation of S-T segment. (See Fig. 15).

Treatment. *Angina pectoris.*—During the attacks, immediate relief is obtained by inhalation of amyl nitrite or by chewing a tablet of nitroglycerine (glyceryl trinitrate) gr. $\frac{1}{200}$ to gr. $\frac{1}{100}$. The patient is advised to carry capsules of amyl nitrite (2 to 5 minims) or tablets of nitroglycerine. As soon as he feels the pain he must stop all exertion and break a capsule of amyl nitrite in a handkerchief and inhale, or chew a tablet of nitroglycerine and keep it under the tongue.

To prevent the attacks, the patient must avoid all exciting causes. If forced to undertake some exertion the patient should be advised to chew a tablet of nitroglycerine beforehand to avoid pain. The general mode of life should be on the lines advised in hypertension. Rest should be determined by the severity of angina and in cases where angina pectoris is repeatedly excited on slight exertion a period of complete bed-rest is advisable. Drugs which dilate the coronary arteries and improve myocardial nutrition are given. Of these, the most effective is *euphyllin* (aminophyllin or theophyllin ethylene diamine) $1\frac{1}{2}$ to 3 grains, 3 or 4 times a day by mouth, or 4 grains intravenously twice a day.

Various surgical procedures have been adopted to abolish the attacks. None of them, however, can cure the cause of the attacks namely the coronary arterial disease and narrowing. They mostly aim at prevention of the pain by interrupting the afferent impulses. as by cervical sympathectomy or paravertebral alcohol injections. Thyroidectomy has been done in some cases with relief of symptoms. Attempts at increasing the blood flow through the myocardium have been made by pericardial suturing of the subpectoral muscle thus establishing new vascular channels. Cardio-omentopexy has also been tried.

Coronary thrombosis.—Treatment is necessarily symptomatic. In acute stage, the pain has to be relieved by morphine gr. $\frac{1}{4}$ subcutaneously. This may be repeated if necessary. For the associated circulatory failure, absolute rest in bed, oxygen inhalation, intramuscular injection of coramine 2 c.c. or cardiazol 1 c.c. are indicated. Purgatives should not be given until the stage of shock is over. If necessary, enema may be given. Later, if congestive cardiac failure develops, treatment with digitalis or other measures detailed in the treatment of congestive failure may be employed. Most important

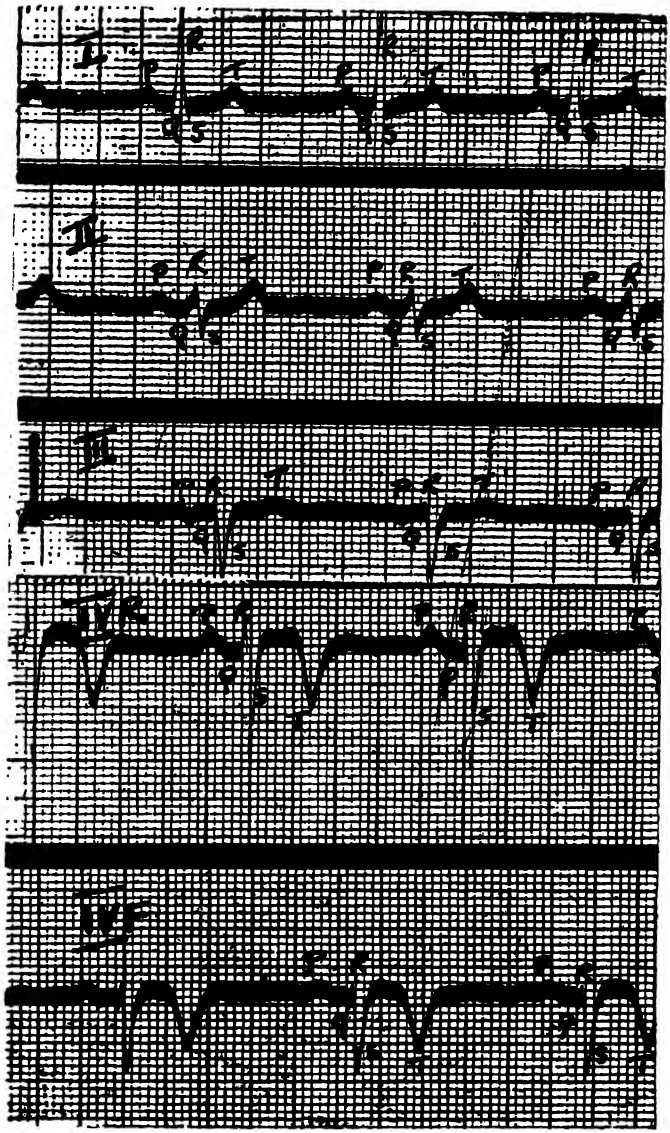


Fig. 15. Coronary thrombosis showing no definite change in the standard leads, but deep inversion of T and elevated S-T segments in the chest leads.

point in the management is ensuring sufficient rest, if necessary with bromides, to allow the formation of a strong scar. In the early stage, patient should not be allowed to exert or sit up in bed at all. At least eight weeks of bed-rest is necessary before allowing the patient up.

Subsequently, the patient should live a restricted life as in angina pectoris. Coronary dilator drugs may also be used to improve the myocardial nourishment.

GRADUAL CORONARY OCCLUSION WITHOUT ANGINA

When small branches of the coronary arteries are gradually obliterated, a slow ischæmic degeneration and fibrosis occur in the myocardium, which ultimately lead to cardiac enlargement and failure. The myocardial fibrosis is chiefly evidenced by electrocardiograph. Thus there may be intraventricular or arborisation block, auriculo-ventricular block, a delay in the intraventricular conduction time or widening of the Q.R.S. complex. Alterations of the S-T. interval, inversion of the T wave and low voltage of the Q.R.S. deflections are also evidences of coronary insufficiency.

CHAPTER VII

CARDIAC AND PULSE IRREGULARITIES

The radial pulse may show two types of irregularity,—(A) irregularity in rhythm, and (B) irregularity in the strength of successive beats.

Irregularities in rhythm.—These again may be of two types.—(1) those with a dominant rhythm, and (2) those without a dominant rhythm.

1. *Irregularities with dominant rhythm.*—(*i*) An otherwise regular pulse may show alternate acceleration and slowing with inspiration and expiration respectively. The cause is sinus arrhythmia.

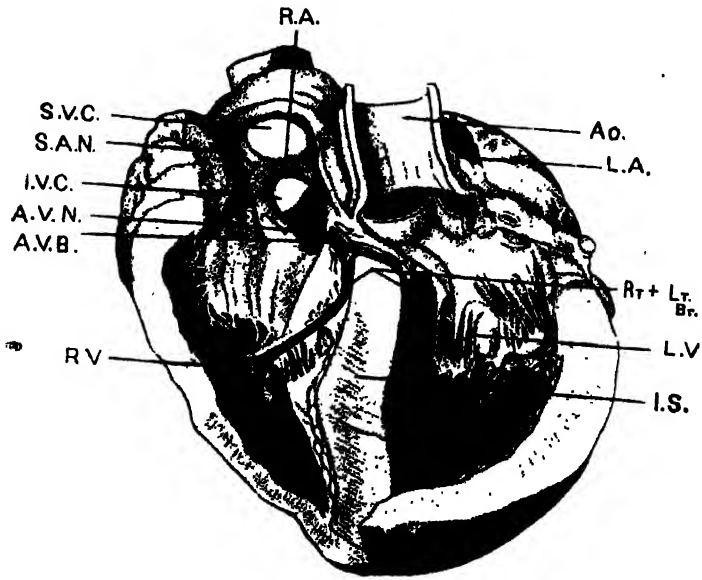


Fig. 16. The conducting system of sheep's heart. R.A.—Right auricle. L.A.—Left auricle. R.V.—Right ventricle. L.V.—Left ventricle. Ao.—Aorta. S.V.C.—Sup. vena cava. S.A.N.—Sino-auricular node. I.V.C.—Inf. vena cava. A.V.N.—Auriculo-ventricular node. A.V.B.—Auriculo-ventricular bundle. Rt. Lt. Br.—Right and left branches of the bundle. I.S.—Interventricular septum.

(*ii*) A regular pulse is disturbed by the occurrence of a premature beat (extrasystole) which is followed by a long pause. This may occur at irregular and long intervals and between the premature beats the rhythm is regular; or the premature beat may occur at regular and frequent intervals dividing the pulse into groups of 2, 3 or more beats separated by long pauses. The pulse beat immediately preceding the long pause is small and occurs prematurely,

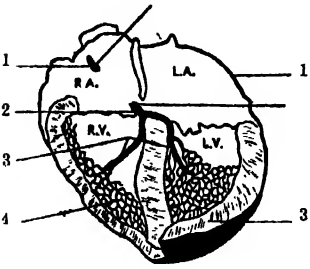
that is, soon after the preceding normal beat. Thus, one type of pulsus bigeminus or trigeminus may be produced.

Disturbances of the pacemaker

1. Sinus tachycardia
2. Sinus bradycardia
3. Sinus arrhythmia
4. Sinus pauses

Disturbances in conduction

1. Sino-auricular block
2. (*a*) Delayed A.V. conduction
   (*b*) Partial heart block
   (*c*) Complete heart block
3. Right or left bundle branch block
4. Arborisation block (intraventricular defective conduction)



Disturbances due to ectopic beats

1. Auricular
   Auricular extrasystole
   Auricular (paroxysmal) tachycardia, flutter, fibrillation
   Nodal
   Nodal extrasystole
   Nodal rhythm
3. Ventricular
   Ventricular extrasystole
   Paroxysmal ventricular tachycardia
   Ventricular fibrillation

Fig. 17. Disturbances of cardiac rhythm and their sites of origin.

(*iii*) A long pause may occur between normal pulse beats in a regular pulse due to auriculo-ventricular block. The ventricle misses a beat due to failure of the auriculo-ventricular bundle to transmit an impulse from the auricles. There is so to speak a 'dropped beat' in an otherwise regular pulse. This again may occur at long and irregular intervals or regularly at short intervals producing pulsus bigeminus, trigeminus etc.

2. *Irregularities without a dominant rhythm.*—The pulse is completely irregular in which no underlying rhythm can be found. The intervals between pulse beats are all unequal, and the successive beats are unequal in strength. This condition is due to auricular fibrillation.

SINUS ARRHYTHMIA

This is an alternate acceleration and slowing of the pulse with respiration, the impulse arising at the S-A node.

This is a normal physiological condition mostly seen in children. Due to excessive vagal tone, the pulse rate is reflexly slowed by expansion of the lung at the end of inspiration. Sometimes, the slowing may be very marked. It always occurs with a slow pulse rate. The irregularity disappears on sympathetic stimulation as on exercise, when the pulse rate is increased. It can be readily diagnosed by a simultaneous record of the respiration and pulse beats. In electrocardiogram, the relation between the P Q R S T waves remains normal.

EXTRASYSTOLE (PREMATURE BEAT)

During the early phase of diastole, the heart muscle is refractory. Towards the later part, however, a cardiac contraction may occur due to an impulse arising from some irritable focus in the myocardium outside the S-A node (normal pacemaker) and is called an extrasystole, or better a premature beat as it occurs before the next normal beat. Immediately after the extrasystole, the myocardium remains refractory for a short time and therefore does not respond to the next normal sino-auricular impulse. The heart remains in diastole until the next normal impulse arrives. There is thus a long pause in the pulse or apex beat which is called the compensatory pause. When the interval between the normal pulse beats preceding and following the extrasystole, equals two cardiac cycles, the compensatory pause is said to be complete. Sometimes, when the pulse rate is very slow and the extrasystole occurs very early in diastole, the myocardium may recover from the refractory state, before the normal impulse from the S-A node arises, so that the normal systole also occurs and there is no compensatory pause, the extrasystoles are then called interpolated. The beat following the compensatory pause is often forcible.

Extrasystoles may be ventricular, auricular or nodal according to the site of origin of the abnormal impulse.

Ventricular extrasystoles.—These are the most common variety and of least importance. The ventricles only show the premature beat, but the auricles are not affected. Immediately after the extrasystole, the auricles contract normally in response to the sino-auri-

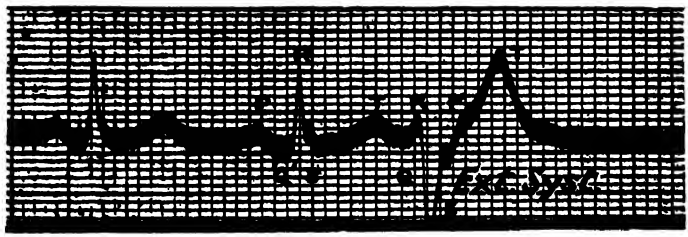


Fig. 18. Electrocardiogram showing ventricular extrasystole.

cular impulse but the ventricles do not respond. The compensatory pause is complete. In venous pulse tracing the extrasystole shows only the *c* and *v* waves. During the compensatory pause an *a* wave occurs which is not followed by *c* and *v* waves. The electrocardiogram shows a premature abnormal Q R S T complex (because of abnormal origin and distribution of the impulse) which is not preceded by a P wave and is followed by a long pause in which P wave alone occurs at the normal time interval after the preceding P wave.

Auricular extrasystoles.—These are less common, but more important, because they are frequently the precursors of auricular fibrillation or flutter and are more often associated with organic heart disease than the ventricular extrasystoles. The premature beat affects both the auricles and ventricles and in the compensatory pause both ventricles and auricles are in diastole; the next normal sino-auricular impulse being inhibited by the general refractory state of the auricular muscle. The compensatory pause however is incomplete because due to the long rest of the sino-auricular node after the premature beat, the next impulse arises a little earlier than when it normally should. In the venous pulse tracing the premature beat shows all the *a c v* waves and is followed by the long pause

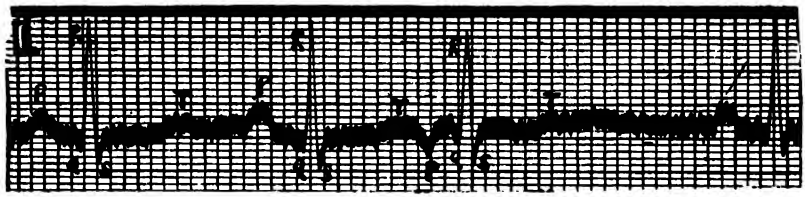


Fig. 19. Electrocardiogram showing auricular extrasystole.

in which no *a* wave occurs. In the electrocardiogram the premature beat shows an inverted P wave, but normal Q R S T waves (although of low voltage). In the long pause there is no P wave and the interval between the P waves preceding and following the extrasystole is not equal to two normal cardiac cycles.

Nodal extrasystole.—This is the rarest variety. The premature impulse arising in the auriculo-ventricular node extends both to the ventricles and the auricles. Both of them contract either simultaneously, or the auricles a little earlier, or later than the ventricles. When the auricles beat earlier than the ventricles the a-c interval of the venous pulse tracing or P-R interval of the electrocardiogram is less than the normal one-fifth second. The P wave is also inverted and may be incorporated in the Q R S complex when the auricles and ventricles contract simultaneously. Q R S complex is normal in character. The compensatory pause is also incomplete.

Aetiology.—Extrasystoles may occur under the following circumstances:

1. In healthy individuals without any organic heart disease commonly excited by fatigue, indigestion, worries, excess of tea and tobacco.

2. In myocarditis due to rheumatic infection or during the course of infective fevers like diphtheria, pneumonia, influenza etc.

3. In myocardial degeneration due to coronary narrowing.
4. Hypertensive heart disease.
5. Digitalis and quinidine poisoning.
6. Chronic rheumatic or syphilitic heart disease.

Symptoms and signs.—Extrasystoles commonly produce a sudden thumping sensation in the chest. When very frequent, there is uncomfortable palpitation. There may be no symptoms at all.

At the radial pulse there is usually a small or weak beat occurring immediately after a normal beat and then a long pause. On auscultation over the heart premature sounds are also audible, preceding the long pause. The recognition of the premature weak beat is important for diagnosis. As mentioned before, the pulse may show occasional extrasystoles at long intervals or there may be pulsus bigeminus or trigeminus. When extrasystole occurs very frequently and irregularly, the pulse may resemble that of auricular fibrillation. In extrasystole, the long intervals are always preceded by a weak and premature beat. Sometimes, the premature ventricular contraction is not sufficiently strong to open the aortic valves, so that there is no premature pulse at the wrist. In such cases, on auscultation over the heart a premature weak first sound will be audible immediately preceding the long pause.

Extrasystoles are generally present with a slow pulse and they tend to disappear when the pulse rate is accelerated by exercise.

Significance of extrasystole.—As a sign of organic heart disease extrasystoles alone are of very little importance. They are more often found without organic heart disease. In the presence of other signs and symptoms of organic heart disease the prognosis will depend on the size of the heart and degree of cardiac failure etc. When such signs are absent extrasystoles may be ignored. During the course of digitalis administration, however, the appearance of extrasystoles is of serious significance and indication of stopping the drug.

Treatment.—Extrasystoles by themselves require no treatment. The associated heart failure, if present, should be treated on the usual lines. In the absence of other signs of organic heart disease, reassurance of the patient is all that is necessary. When there is anxiety and discomfort sedatives like potassium bromide should be given. When extrasystoles are very frequent causing severe discomfort, quinidine gr. 3, three times a day may be given to stop or reduce the extrasystoles.

HEART-BLOCK

Three varieties of heart-block may occur.—1. Sino-auricular block. 2. Auriculo-ventricular block. 3. Intraventricular or bundle-branch block.

Sino-auricular block.—It is an arrhythmia due to a depression of the S-A node. The heart as a whole is slow or misses a beat. During the long pause there is no contraction of either the auricles or the ventricles as seen in the electrocardiogram. This is due to increased vagal tone.

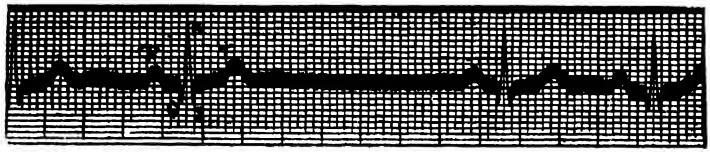


Fig. 20. Electrocardiogram showing sino-auricular block.

Auriculo-ventricular block.—This is the type commonly referred to as heart-block. The ventricles fail to respond to the auricular beats due to a failure of the auriculo-ventricular bundle to conduct the impulses from the auricles to the ventricles. The failure of conduction may be of three degrees, as follows:—

(*a*) A delay in the conduction, but all the impulses from the auricles eventually reach the ventricles. The normal conduction time of one-fifth second is exceeded as can be recognised by the venous pulse tracing or the electrocardiograph.

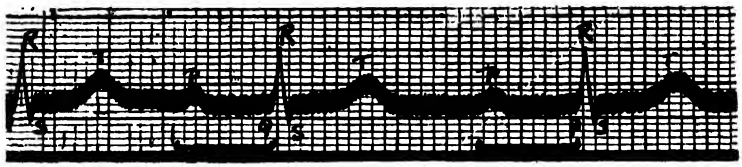


Fig. 21. Electrocardiogram showing prolonged P-R interval (auriculo-ventricular conduction delay).

(*b*) Incomplete block, in which some of the auricular impulses fail to reach the ventricles. The ventricles, therefore, miss some of the auricular beats and long pauses occur in the pulse. The pauses may occur at long and irregular intervals when an occasional auricular impulse is blocked. In higher grades of block, every fourth or third auricular beat may fail to reach the ventricles, and therefore a long pause follows every 3 or 2 ventricular beats and pulsus trigeminus or pulsus bigeminus occur. In still higher grades of block, every second or third or fourth auricular impulse is transmitted and a very slow but regular pulse occurs. The ratio of the auricular and the ventricular beats in various grades of incomplete heart-block may thus be 4:3, 3:2, 2:1, 3:1, 4:1 etc.

(*c*) Complete block.—None of the auricular impulses are conducted by the auriculo-ventricular bundle. There is a temporary standstill of the ventricles and then impulses are set up either in the ventricles or in auriculo-ventricular node at an independent and slow rhythm.

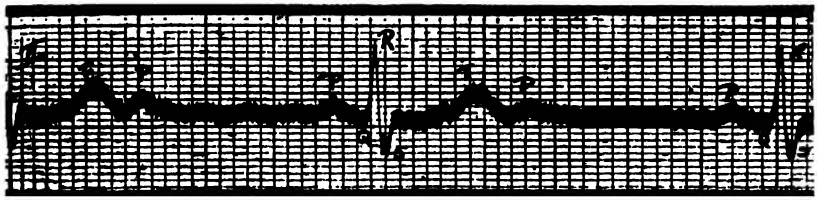


Fig.22. Electrocardiogram showing incomplete heart block. Auriculo-ventricular ratio 2: 1.

Thus, an idioventricular or nodal rhythm is established. In idioventricular rhythm, the ventricles and the auricles beat independent of each other. In nodal rhythm, both auricles and ventricles respond to the nodal impulse.

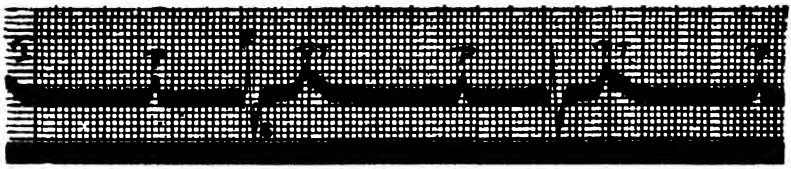


Fig. 23. Electrocardiogram showing complete heart-block. The auricles (P waves) and ventricles (QRST complexes) have independent rhythm.

Aetiology.—Heart-block may occur in:—

(*a*) Acute myocarditis due to rheumatic infection, diphtheria, pneumonia, influenza etc.

(*b*) Intoxication by digitalis, quinidine, strophanthus.

(*c*) Myocardial degeneration and fibrosis due to coronary insufficiency with or without hypertension, or in myocardial infarction.

(*d*) Syphilis producing gummatous involvement of the bundle—a rare cause contrary to the usual view.

(*e*) Congenital interventricular septal defect.

Heart-block is more often due to a functional depression of auriculo-ventricular bundle than to any demonstrable structural change as in the last three conditions mentioned above.

Symptoms.—There may be no symptoms in heart-block. When however the long pauses are sufficiently long to produce cerebral anæmia, patient experiences various degrees of syncope from slight giddiness to unconsciousness with convulsions. Adams-Stokes syndrome described before (see page 40) is most likely to occur

when temporary cardiac standstill occurs during transition from an incomplete to a complete block.

Sometimes, the strong ventricular beat, immediately following the long pause due to ventricular overfilling, produces a throbbing sensation in the chest or neck.

Signs.—As mentioned before, the pulse may show occasional dropped beats at irregular intervals or the pulse may be regularly irregular showing pulsus bigeminus, trigeminus etc. The long pauses however are not preceded by any premature beat at the wrist or premature sounds at the cardiac apex as in extrasystole. During the long pause, though no pulse is felt at the wrist, a venous pulse in the jugular veins may be seen due to auricular contraction.

When a 2:1 rhythm is established with a normal auricular rate, the pulse is regular but very slow; thus with auricular rates varying from 72 to 80 per minute, pulse rates between 36 to 40 per minute are obtained. In the jugular vein, the venous pulse will show a rate double that at the wrist. Still higher grades of block generally occur with rapid auricular rates as in flutter and fibrillation.

In complete heart-block with idioventricular rhythm the pulse rate is often 36 to 40 per minute though sometimes it may fall below 20. In nodal rhythm, the pulse rate is generally about 40 per minute. In both idioventricular and nodal rhythm, the pulse rate shows no response to exercise. In incomplete block, there may be sudden changes in pulse rate due to changes in the grade of block by exercise.

In the sphygmographic tracing, the long pauses are preceded and followed by normal pulse beats. In the venous pulse during the long pauses in incomplete block *a* waves occur alone without any *c* and *v* waves. In the electrocardiogram, all the P waves are not followed by ventricular complexes. (See Fig. 22).

In complete heart-block, while the radial pulse tracing shows a regular slow rhythm, the venous pulse shows complete dissociation between the *a* waves and the *c* and *v* waves. In electrocardiogram also P waves and ventricular complexes are independent of each other. The latter may also be of abnormal shape. (See Fig. 23).

Significance of heart-block.—Unlike extrasystole, heart-block always indicates some organic involvement of the myocardium either toxic or structural. It is therefore, of more serious significance. Higher degrees of block or complete block are indications of severe involvement. In minor degrees of block the prognosis should be based on other signs such as size of the heart, exercise tolerance or severity of cardiac failure. Adams-Stokes syndrome is of bad prognosis if it occurs frequently. Death may occur during an attack from cardiac standstill.

Treatment.—Treatment should be directed mainly towards the underlying organic heart disease which is present. If cardiac failure is present, this should be treated.

For the heart-block itself, the conductivity of the auriculo-ventricular bundle may be improved by injections of adrenaline or atropine, or by ephedrine by mouth. In severe cases with repeated syncopal attacks or Adams-Stokes syndrome repeated injections of adrenaline may be necessary. During an attack of syncope with cardiac standstill, adrenaline 1 in 1000, ½ c.c. should be given intravenously and the vein massaged towards the heart. It may also be given intracardiac. To prevent attacks, ephedrine hydrochloride gr. ½ every 4 hours by mouth or atropine sulphate gr. $\frac{1}{100}$ subcutaneously every 6 hours should be given. Barium chloride gr. ½, 3 times a day may also be tried to prevent heart-block.

Intraventricular block (Bundle-branch block, Arborisation block).—This is a defective conduction of the cardiac impulse in one of the main branches of the auriculo-ventricular bundle *(Bundle-branch block)* or in its wide ramifications *(Arborisation block)*. This condition cannot be recognised clinically because there is no disturbance in rhythm. Occasionally, the impulse reaching one of the ventricles a little later than the other, causes their asynchronous contraction and the first sound may be reduplicated

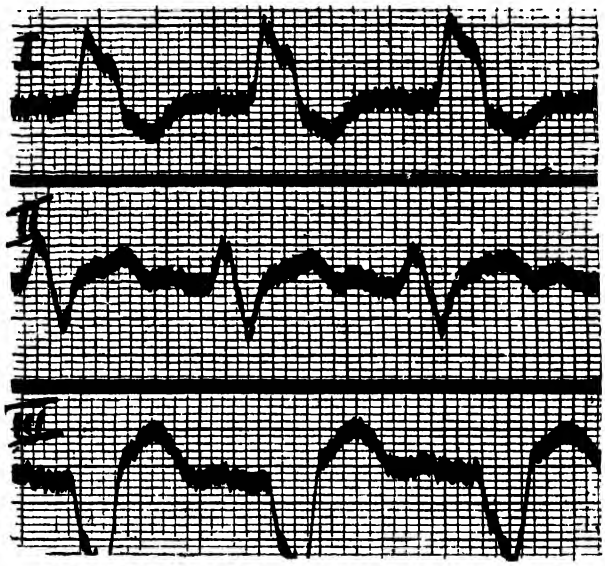


Fig. 24. Electrocardiogram showing bundle-branch block Type I.

This condition is however mostly recognised by electrocardiograph. The Q R S complex is wider (longer than normal 0·1 sec), R wave is notched and T wave is deep and in a direction opposite to

the main deflection R or S. Two types of curves are seen. In Type I (Left bundle branch block) the main initial deflections of R and S resemble that of left axis deviation—maximum deflection of Q R S complex upward in lead I and downward in lead III and in Type II (Right bundle branch block), they resemble right axis deviation —maximum deflection of Q R S complex downwards in lead I and upward in lead III.

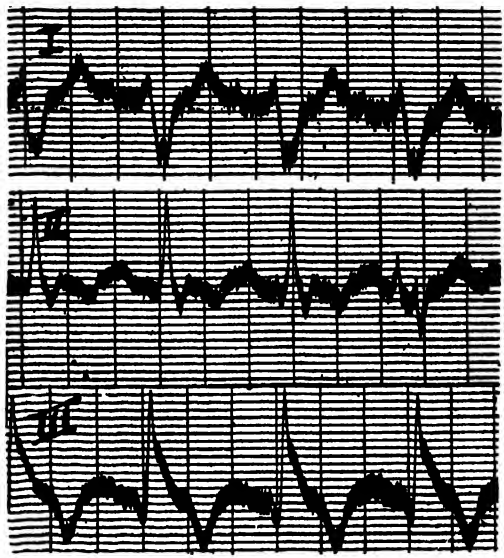


Fig. 25. Electrocardiogram showing bundle-branch block Type II.

Causes.—The causes of intraventricular block are 1. coronary atheroma with or without hypertension, 2. acute myocarditis of rheumatic infection, diphtheria etc., 3. digitalis or quinidine poisoning etc.

Significance.—It is of serious significance as it indicates widespread myocardial damage or severe toxic effect.

AURICULAR FIBRILLATION AND FLUTTER

These are conditions in which the auricular systole is replaced by a more or less continuous fibrillary twitching in individual muscle fibres at a very rapid rate. This is due to the establishment in the auricular muscle of a wave of excitation and contraction travelling at a very high rate along a more or less circular pathway, usually around the openings of the large veins. This wave will persist so long as it finds responsive tissue ahead, that is, when the

velocity of the wave and the length of the circular path are such that it reaches the starting point after the refractory state following the previous contraction has disappeared. This is called the *circus movement.* In auricular fibrillation, the path is wider and irregular and rate of the wave is very rapid, varying from 300 to 600 per minute. In flutter however, the path is narrower and regular, and the rate is slower between 200 to 400 per minute.

Effect of fibrillation and flutter on the heart and circulation. On the auricles.—As mentioned before, the auricular systole is lost. This however does not interfere with the ventricular filling which is accomplished by venous pressure and gravity. All the phenomena associated with auricular systole however are lost. Thus, the presystolic murmur of mitral stenosis disappears with the onset of auricular fibrillation. In the venous pulse tracings, the *a* waves disappear and in the electrocardiogram, P waves are absent. Irregular small oscillations at very rapid rate however are seen in the electrocardiogram.

On the ventricles.—Impulses at a very rapid rate reach the auriculo-ventricular bundle for conduction. A functional block is established because of the inability of the bundle to transmit impulses at such a rapid rate. In auricular flutter, the impulses reach the auriculo-ventricular bundle at a regular rate and a definite ratio being established between the auricular and ventricular rates the latter shows a rapid but regular rhythm. In auricular fibrillation however, the impulses are very irregular and of varying strength, so that the ventricular rate is very irregular; the contractions also vary widely in strength. Some of the weaker contractions may fail to open the semilunar valves (frustrane contractions.) The effect on the ventricles therefore, is one of increase in rate and thus increase of work. Moreover, there is waste of energy in auricular fibrillation due to frustrane contractions. If the ventricular myocardium is healthy this increase of work may not be of any importance and is well tolerated. If however, ventricles are already damaged; this will precipitate failure or aggravate it when present.

Aetiology.—Auricular fibrillation is commonly seen in the following conditions:

1. Rheumatic carditis with mitral stenosis. 2. Thyrotoxicosis. 3. Hypertensive heart disease. 4. Coronary atheroma. 5. Acute myocarditis of infectious fevers. 6. Focal sepsis, e.g. apical dental abscesses. 7. In apparently healthy people without any obvious cause.

Auricular flutter is much less common than fibrillation and may be seen in the same conditions. It is more often paroxysmal in nature.

Symptoms.—A sensation of fluttering and palpitation are the only symptoms directly due to fibrillation or flutter. Commonly however, the associated symptoms of heart failure predominate.

Signs. *Fibrillation.*—Pulse is irregularly irregular and no dominant rhythm is present. The rate varies from 90 to 160 per minute. There is increase of pulse rate with exercise with increase of irregularity. The pulse beats vary in strength and there is no sequence of a long pause following a small and premature beat as in extrasystole. Some of the ventricular contractions being too weak to produce a pulse, the ventricular rate at the apex does not correspond with the pulse rate at the wrist (a condition of pulse deficit). The pulse tracings show complete irregularity.

The venous pulse shows an absence of the *a*-waves, and clinically the large waves in the jugular veins are absent.

In the electrocardiogram, ventricular complexes are very irregular in rhythm and voltage and are not preceded by P waves. Irregular and rapid small auricular oscillations are seen. (Fig. 26).

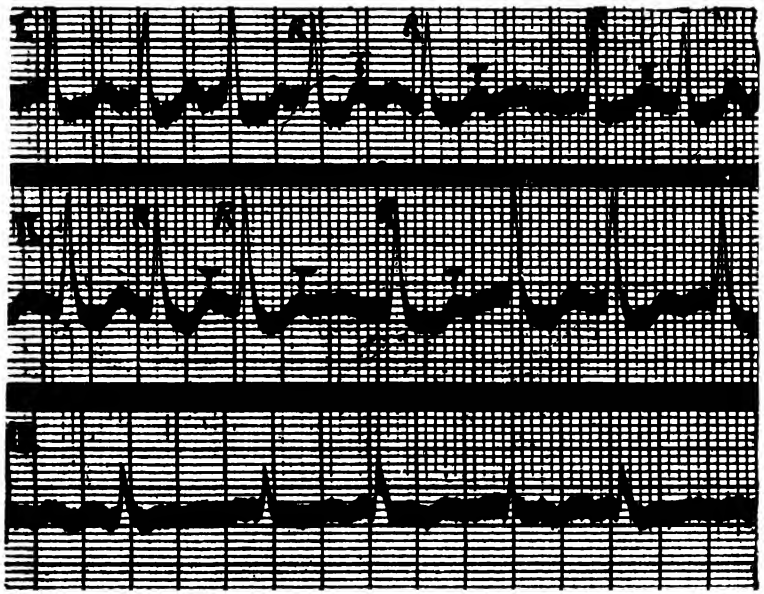


Fig. 26. Electrocardiogram showing auricular fibrillation.

Flutter.—The pulse is regular both in rhythm and strength of successive beats. Rate varies from 140 to 180 per minute. On exercise there is no increase of pulse rate. Carotid pressure may cause sudden changes in pulse rate due to variation in the grade of block.

In the electrocardiogram small rapid but regular auricular oscillations are seen and the ventricular complexes occur regularly bearing a ratio with the auricular waves which are usually inverted. (Fig. 27).

Course and prognosis.—Fibrillation once established tends to be permanent. In the early stages however, it may occur in paroxysms. Fibrillation is usually associated with organic heart disease and the prognosis in such cases depends on the myocardial efficiency and the ætiology. Even with impaired myocardium, auricular fibrillation

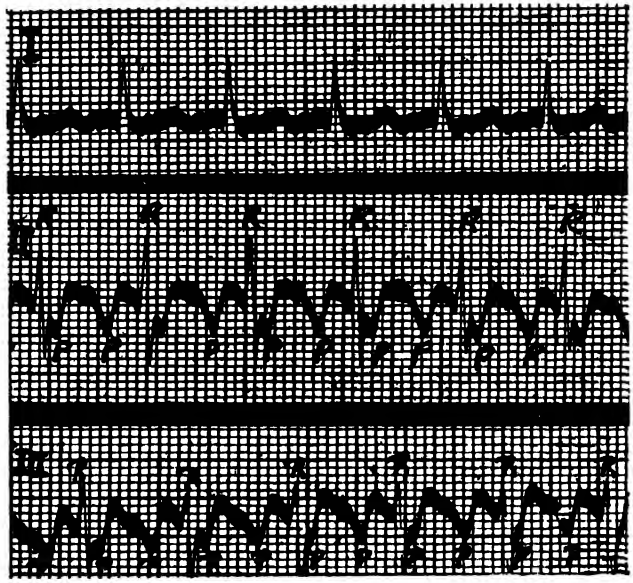


Fig. 27. Electrocardiogram showing auricular flutter.

does not increase the seriousness if the ventricular rate can be controlled within normal limits by digitalis. When the cause is removable, as in thyrotoxicosis, prognosis is good.

Treatment.—When the auricular fibrillation complicates congestive cardiac failure, the treatment consists in controlling the ventricular rate by digitalis. It is in such cases, that digitalis therapy is most successful in causing rapid improvement. The ventricular rate should be maintained between 60 to 70 beats per minute with continuous maintenance doses of digitalis. Digitalis has no effect in stopping the circus movement. Flutter is however, changed by digitalis into fibrillation and when digitalis is stopped, normal rhythm may return.

In cases of recurrent auricular fibrillation where no signs of congestive failure are present, attempt may be made to stop the circus movement by quinidine. Effect of quinidine is to increase the refractory state of the myocardium and also to reduce its conductivity. If the latter balances the former, it will not succeed in stopping the circus movement.

Quinidine has the following disadvantages:

1. It does not succeed in all cases. Even when it succeeds in establishing normal rhythm, fibrillation recurs in the majority of cases.

2. Because of its severe depressing action on the myocardium, there may be sudden death from cardiac standstill specially in the presence of gross organic disease of the myocardium.

3. Establishment of normal auricular systole may dislodge intra-auricular thrombi and cause embolism.

4. Some patients show idiosyncracy to quinidine.

Quinidine is contraindicated in the presence of enlargement of the heart, cardiac failure and in fibrillation of more than 6 months' duration.

Dosage of quinidine.—A small dose of 3 grains should be first given to test idiosyncrasy. If there is no idiosyncrasy, 5 grains should be given 3 times a day on the first day, 4 times on the second day and 6 times on the third day and then it should be gradually decreased in successive days. During this course, the patient should preferably be in bed rest and a watch is kept on his pulse rate and for toxic symptoms. These are headache, tinnitus, nausea, vomiting, sweating, abdominal pain, and purpura. Dangerous symptoms are dimness of vision and ventricular extrasystoles. If possible, electrocardiographic records to determine auricular rate should be taken and the drug discontinued if auricular rate falls below 250 per minute.

When normal rhythm is established, a maintenance dose of 5 grains daily may be given to prevent a relapse.

PAROXYSMAL TACHYCARDIA

This is a condition of sudden onset and offset of tachycardia lasting for a variable duration and due to production of waves of excitation and contraction at a very rapid and regular rate from some point in the myocardium outside the normal pacemaker. According to the site of origin, paroxysmal tachycardia may be auricular, ventricular and nodal. The site of origin can only be determined by electrocardiograph.

In auricular variety inverted P waves occur at very rapid rate (160 to 180 per minute) and are followed by ventricular complexes also at the same rate. In ventricular variety, the Q R S complexes are of abnormal shape like those of premature beats. P waves occur at regular and normal rate. The nodal variety is least common and they resemble the nodal premature beats but occurring at very rapid succession.

The ætiology of paroxysmal tachycardia is like that of extrasystoles. They are more often found in apparently healthy people without any other sign of organic heart disease. The auricular

variety is more common and is less often associated with organic heart disease than the ventricular variety which is of more serious significance.

Symptoms.—Palpitation is the commonest symptom. Sometimes, the extreme rapid ventricular rate interferes with ventricular filling, and cardiac output being diminished, syncope may occur. Anginal pain or symptoms of rapidly developing cardiac failure may occur with dyspnœa, venous congestion and œdema specially in those with organic heart disease.

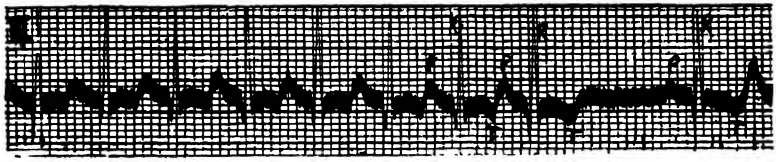


Fig. 28. Electrocardiogram showing auricular paroxysmal tachycada. (The end of a paroxysm).

Signs.—Pulse is very rapid, usually between 160 to 180 or more per minute. It is regular and does not respond to exercise or rest. Carotid pressure or pressure on eyeballs may suddenly stop the paroxysm. Other signs depend on the presence of organic heart disease. Cardiac enlargement, signs of congestive failure, gallop rhythm, tic-tac rhythm, pulsus alternans etc., may appear due to ventricular exhaustion.

Course and prognosis.—The paroxysm may last from a few seconds to several hours. Occasionally, it lasts for days or even months. Frequency of attacks also varies. Its effect on the heart depends on the condition of the myocardium. In healthy people without any obvious organic heart disease it is of little significance. In the presence of organic heart disease however, it precipitates failure.

As a sign of organic heart disease, paroxysmal tachycardia alone is of very little importance like extrasystole. The ventricular variety however, is more often associated with organic disease and so of more serious significance.

Treatment.—During a paroxysm, the patient should be at rest. In shorter attacks, reassurance and sedatives like bromides are sufficient. The attack stops spontaneously. Various physical measures are adopted to stop the paroxysm by inducing reflex vagal stimulation, such as stooping forwards with the head low, pressure on the carotid sinus or the eyeballs and pulling out the tongue. Induction of vomiting by large doses of tincture of ipecac may also succeed. In prolonged attacks, the following drugs may be used. Digitalin gr. $\frac{1}{100}$ or Digalen 0.1 to 0.5 gm. intravenous, or strophanthin gr. $\frac{1}{240}$ intravenous. Digoxin 0.5 mg. in 1 c.c. diluted with 9 c.c. of

normal saline intravenously may be tried. More recently, an acetyl cholin derivative, Carbachol or Doryl in 1 to 2 mg. doses 3 times a day by mouth has been used with success. Quinidine in usual doses may also be tried.

Digitalis and quinidine may be continued to prevent recurrences.

Irregularities in strength of successive beats with regular rhythm. 1. *Pulsus alternans.*—This is a condition of alternate strong and weak pulse beats due to a severe weakness or exhaustion of the left ventricle. When marked it can be easily recognised by palpation of the radial pulse. It can be easily recognised by sphygmographic tracing and by the sphygmomanometer. With the latter at or near the systolic pressure the pulse becomes half and on lowering the pressure further the weaker beats appear.

It is usually a serious sign of left ventricular failure and is of bad prognosis. It may however, occur temporarily in paroxysmal tachycardia, the myocardium being exhausted by the excessive rapid rate; recovery follows the cessation of the paroxysm.

2. An interpolated extrasystole occurring after each normal beat may cause a *pseudo alternans* pulse.

CHAPTER VIII

Pericarditis may be of the following types.—1. Acute fibrinous pericarditis. 2. Pericarditis with effusion—(*a*) Serofibrinous. (*b*) Purulent and (*c*) Hæmorrhagic. 3. Chronic pericarditis—(*a*) Chronic constrictive pericarditis and (*b*) Chronic mediastino-pericarditis.

ACUTE FIBRINOUS PERICARDITIS

Aetiology.—(*a*) Rheumatic infection. (*b*) Pneumonia. (*c*) Tuberculosis. (*d*) Septicæmias. (*e*) Coronary thrombosis. (*f*) Uræmia.

In the last two conditions, the inflammation is non-infective and possibly toxic in cases of uræmia. In others the common organisms are D. pneumoniæ, Streptococcus hæmolyticus, Staphylococcus aureus etc. In rheumatic infection, no particular organisms can be isolated.

Pathology.—A thick fibrinous deposit forms on the pericardium which becomes shaggy in appearance due to constant movement. The appearance is described as *bread-and-butter heart.*

Symptoms and signs.—There may be no symptoms. Pain is complained of when the pleuro-pericardial surface is particularly affected. A pericardial rub is the characteristic finding. It is commonly heard at the base of the heart or along the left border of the sternum.

PERICARDITIS WITH EFFUSION

(*a*) **Serofibrinous. Aetiology.**—The causes are practically the same as in fibrinous pericarditis except uræmia and coronary thrombosis. Besides, it may occur in polyserositis (Concato's disease), in which serous effusion in pericardial, pleural and peritoneal cavities occurs without any obvious cause.

Pathology.—The serous effusion gradually distends the pericardium which may reach enormous size. Pressure effects are seen on the mediastinal structures, the big veins, specially the inferior vena cava and hepatic veins with consequent passive congestion of the liver. Compression of the heart interferes with the diastolic filling, and blood pressure and pulse pressure fall. In large effusions, the left lung is compressed and collapses against the posterior chest-wall.

Symptoms and signs.—It generally occurs as a complication in the course of an acute rheumatic fever, pneumonia or septicæmic conditions. Its onset is marked by a further rise of temperature. When effusion is rapid with compression of veins, dyspnœa is common.

The signs are (*a*) Præcordial bulging or fullness. (*b*) Extension of cardiac dullness beyond the apical impulse which may be displaced upwards. (*c*) Pear-shaped area of dullness in erect posture. (*d*) Rotch's sign (see page 20). (*e*) Engorgement of neck veins. (*f*) Enlarged liver. (*g*) Pulsus paradoxus (see page 28). (*h*) Ewart's sign—a patch of dullness, bronchial breathing and bronchophony at the angle of the left scapula.

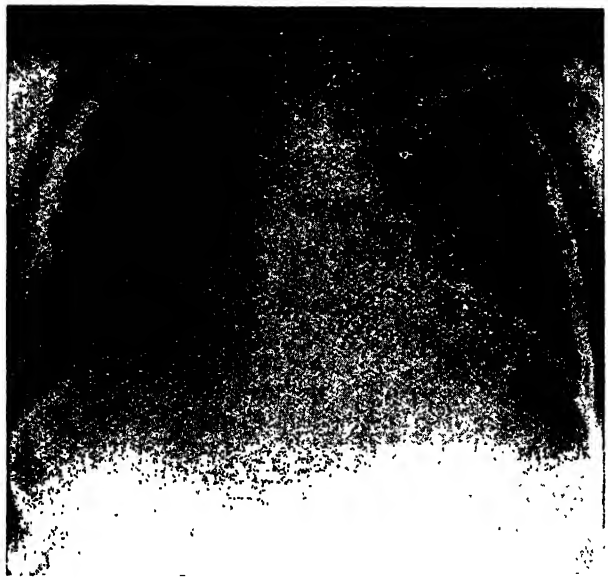


Fig. 29. Pericardial effusion as seen under X-rays.

Diagnosis.—Diagnosis is confirmed by fluoroscopy or skiagram and by paracentesis. The condition may be confused with cardiac enlargement, mediastinal growths and aortic aneurysm.

(*b*) **Purulent pericarditis.**—This is produced by direct spread of infection from a neighbouring suppurative focus like empyema, lung abscess etc., or by blood-borne infections as in septicæmias. The organisms are mostly Streptococcus hæmolyticus, Staphylococcus aureus or D. pneumoniæ.

The signs and symptoms are those of effusion. The nature of the fluid is diagnosed by paracentesis.

(*c*) **Haemorrhagic effusion.**—This may occur in malignant infiltration of the pericardium, in injuries or in tuberculous infection.

Course and prognosis.—Acute fibrinous and serofibrinous pericarditis usually complicates acute rheumatic infection or pneumonia or other infections. The prognosis depends on the severity of these

diseases. Pericarditis may only be a terminal condition in uræmia, or a passing complication in the course of rheumatic infection, pneumonia, tuberculosis, coronary thrombosis etc. Purulent pericarditis is however of serious prognosis. A very large effusion may cause sufficient embarrassment of cardiac function to require relief by paracentesis. Chronic pericarditis may follow with gradually increasing fibrosis and adhesions with their consequences.

Treatment.—In most cases, the treatment consists in the symptomatic relief besides the measures taken for the eradication of the primary disease.

Pain if present should be relieved by sedatives and analgesics.

In pericardial effusion, if there is evidence of excessive pressure-effects, relief by paracentesis is indicated. Paracentesis is also indicated in all cases of purulent effusion. Aspiration can be done by a large bore needle attached to a 50 or 100 c.c. syringe or if necessary by the Potain's aspirator very carefully. The site of puncture is the fifth intercostal space a little internal to the outermost point of dullness.

Chemotherapy to control the infection in cases of pneumococcal, streptococcal or staphylococcal infections with sulphanilamide, sulphathiazol etc., is also indicated.

CHRONIC PERICARDITIS

This generally follows acute pericarditis when organisation of the fibrinous exudate leads to fibrous thickening of the pericardium and adhesions between its layers. Such thickening and adhesions may vary in degree from slight localised patches (*milk spots*) on the visceral pericardium to extensive thickening and adhesions between the two layers of pericardium and with the surrounding structures such as the chest wall, diaphragm etc.

Pathology.—Slight thickening of the pericardium or adhesions between the two layers do not interfere with cardiac function and are unimportant. When however, there are adhesions with the surrounding mediastinal structures anchoring the heart to the chest wall, diaphragm etc., increase of work for the heart with consequent hypertrophy and later failure may occur. This condition is called *chronic mediastino-pericarditis.*

Sometimes, the pericardium all over the heart is adherent and undergoes progressive thickening and even calcification. Such a tough and unyielding covering causes a chronic compression of the heart and interferes with its diastolic filling as well as systolic contraction. The thickened pericardium may contract and cause obstruction specially at the openings of the superior and inferior venæ cavæ, and even the hepatic veins. This condition is called *constrictive pericarditis.* Venous obstruction to the hepatic veins and inferior vena cava causes a passive congestion of the liver which

may ultimately show cirrhosis. The condition is described as *pericardiac pseudocirrhosis of liver* or *Pick's disease*.

Aetiology.—Chronic pericarditis may be due to tuberculosis, Concato's disease (*Poly-serositis*), pneumonia, rheumatic infection, and other infections. Sometimes, there is no obvious cause. In chronic constrictive pericarditis, evidence of rheumatic infection is less obvious.

Symptoms and signs.—There may be no symptoms or signs in slight pericardial thickening.

Mediastino-pericarditis.—Symptoms appear when there is congestive cardiac failure. Dyspnœa is the commonest symptom.

Signs.—1. Systolic retraction of the præcordium and lower intercostal spaces; characteristically in the lateral and posterior chest wall (*Broadbent's sign*). 2. Cardiac hypertrophy and enlargement. 3. Position of apical impulse does not move with change of position of the patient.

Chronic constrictive pericarditis.—Symptoms are dyspnœa, abdominal enlargement and œdema of ankles.

Signs.—1. Engorgement of cervical veins without pulsation. 2. Ascites. 3. Enlargement of liver. 4. Heart is of normal size and shows no definite valvular lesions. Apical impulse indistinct or absent. 5. Pulse is small in volume and low in tension. 6. Pulsus paradoxus. 7. X-ray shows borders of the heart straighter and pulsations are restricted. Calcification may be seen in pericardium. 8. Electrocardiogram shows low voltage of Q R S complex and T may be inverted.

Prognosis.—Slight pericardial adhesions or thickening which do not increase the work of the heart or interfere with venous return are of no importance. Constrictive pericarditis or mediastino-pericarditis however, causes chronic invalidism and death from cardiac failure unless relieved by surgery.

Treatment.—Surgical treatment is the only hope for permanent benefit. Medical treatment for congestive failure with rest, diuretics etc., may cause temporary improvement.

For mediastino-pericarditis, thoracolysis or rib removal (*Brauer's operation*) and for constrictive pericarditis, cardiolysis or removal of præcordial ribs and left half of lower sternum, cutting away of the thickened pericardium over the heart and constricting bands over the inferior vena cava (*Delorme's operation*) are indicated.

CHAPTER IX

CONGENITAL HEART DISEASE

Various developmental errors in the heart may occur, but only those which allow a free entry of the venous blood into the arterial system or throw a great strain on the cardiac muscle are of importance. When communication between the right and the left side of the heart exists there is usually an arterio-venous shunt, that is, blood flows from the arterial to the venous side because of higher pressure in the former. There is some increase of work for the right heart which hypertrophies but no symptoms appear as in cases of patent interventricular septum or mild cases of patent ductus arteriosus. If however, there is a veno-arterial shunt, that is, flow of blood from venous to the arterial side as in interventricular septal defect with a dextro-position of the aorta and pulmonary stenosis, cyanosis will be present.

Classification.—Congenital heart diseases are classified according to the presence or absence of cyanosis into three groups as follows:

1. *Acyanotic.*—No communication exists between the right and the left side of the heart. Some of these conditions are harmless, others may cause heart failure, sooner or later and some are incompatible with life.

(*a*) *Simple dextrocardia.*—No symptoms and harmless.

(*b*) *Coarctation of aorta.* (*i*) *Infantile type.*—Narrowing of the aorta between the left subclavian artery and the point of entry of the ductus arteriosus. Duration of life is very short.

(*ii*) *Adult type.*—Narrowing of the aorta just at the junction with the ductus arteriosus. Duration of life is longer.

(*c*) *Subaortic or aortic stenosis.*—Duration of life is short.

(*d*) *Ectopia cordis.*—Extra thoracic heart. Not compatible with life beyond a few days after birth.

2. *Potential cyanotic group.*—Communication between venous and the arterial side exists, but there is usually arterio-venous shunt. When right ventricular failure occurs with increase of pressure on the right side of the heart veno-arterial shunt is established.

(*a*) *Patent ductus arteriosus.*—Compatible with long life. May terminate with right heart failure or subacute bacterial endocarditis.

(*b*) *Patent interventricular septum.*—No shortening of life.

(*c*) *Patent foramen ovale.*—No shortening of life.

(*d*) Communication between the base of the aorta and pulmonary artery. Patient may reach adult age.

3. *Cyanotic group.*—A veno-arterial shunt exists from the beginning.

(*a*) *Tetralogy of Fallot.*—Pulmonary stenosis, patent interventricular septum, dextroposition of the aorta and right ventricular hypertrophy. Duration of life is short, but patients may reach adult life.

(*b*) Pulmonary stenosis with patent foramen ovale.

(*c*) Patent interventricular septum with dextroposition of the aorta. May reach adult life.

(*d*) *Cor triloculare biatrium.*—Duration of life short.

(*e*) *Transposition of the arterial trunk.*—Short duration of life.

There may be other anomalies and multiple defects are common.

Symptoms.—There may be no symptoms at all. Symptoms of heart failure, such as dyspnœa on exertion or hæmoptysis from pulmonary congestion may occur. Epistaxis and cerebral symptoms of defective circulation may occur in cyanotic group due to polycythæmia.

Signs.—The most prominent signs are seen in the cyanotic group. Marked cyanosis, known as *morbus caeruleus,* is common. Other signs are clubbing of the fingers, stunted growth, both mental and physical, polycythæmia and signs of venous congestion when congestive failure is present e.g., œdema, enlarged liver and engorged neck veins.

Cardiac signs depend on the nature of the defect. Right ventricular hypertrophy and enlargement are common. Systolic murmur and thrill are also common. Murmurs may be completely absent even with serious defects like trilocular heart.

Special signs in individual defects have been discussed in the case-taking chapter.

Complications.—The common complications are congestive heart failure, bacterial endocarditis and secondary infections of the lung.

CHAPTER X

BACTERIAL ENDOCARDITIS

INFECTIVE, ULCERATIVE OR MALIGNANT ENDOCARDITIS

This is a condition of bacterial infection of the cardiac valves with formation of vegetations, which contain large number of micro-organisms.

There are two varieties,—acute and subacute. The main difference between the two lies in the virulence of the infecting organisms and the clinical course, the acute type being fatal in six weeks.

Infecting organisms and their source.—*In acute bacterial endocarditis,* the commonest micro-organism is Streptococcus hæmolyticus. Less commonly, D. pneumoniæ, Staphylococcus aureus, N. gonorrhœæ and N. meningitidis are found. The source of infection is some obvious focus of sepsis such as the puerperal uterus, osteomyelitis, pneumonia and skin infections like boils, carbuncles etc.

In subacute bacterial endocarditis, Streptococcus viridans is the most constant micro-organism. Very rarely Pfeiffer's bacillus is found. The source of infection is not obvious.

Predisposing factors.—In most cases, the vegetations develop on previously damaged or deformed valves. These are mostly rheumatic valvular lesions or congenital deformities such as, rheumatic mitral stenosis or aortic regurgitation, congenital bicuspid aortic valves, pulmonary stenosis, patent ductus arteriosus etc.

In acute type however, infection may occur on healthy valves.

Pathology. *Heart.*—Vegetations which are sometimes of very large size are seen on the cardiac valves. The left side of the heart, that is, the mitral and the aortic valves are mostly involved. In *subacute variety,* the vegetations are larger, more tough, less destructive and show extension on to the mural endocardium, chordæ tendinæ and the base of the aorta. The vegetations consist of fibrin, platelets and micro-organisms. There are very few polymorphonuclear cells. In the deeper portions fibroblastic proliferation is seen and in older vegetations, fibrosis and often calcium deposition may be seen. In *acute variety,* the vegetations are more friable and destructive. The valves may be perforated or torn. The myocardium may show cloudy swelling and in acute cases focal suppuration. Coronary arteries may get blocked by emboli from the vegetations.

Spleen.—It shows enlargement and infarction, sometimes multiple infarcts.

Kidneys.—These show enlargement with petechial spots on the surface. Large infarcts may also occur. Microscopically, an embolic focal glomerulonephritis is common. Rarely, diffuse glomerulonephritis with renal failure may occur.

Embolism and infarction in various organs due to torn fragments of vegetations being dislodged in the circulation may occur. In acute type, the infarcts may suppurate.

Symptoms and signs. *Acute bacterial endocarditis.*—The onset is sudden with rigor. In cases when it develops during the course of pneumonia or a septic infection elsewhere, the onset may be marked by a further rise of temperature, appearance of rigors and sweating, pain and swelling in joints, etc. Sometimes the disease is not revealed until some embolic manifestations occur. The signs are those of a septicæmia with septic type of fever, severe illness with toxæmia, petechial or purpuric hæmorrhages in skin, conjunctiva and mucous membranes, pain and swelling of joints. No cardiac signs may be present or new murmurs of more recent damage may appear. Some enlargement of the heart may occur.

There is a high leucocytosis with increase of polymorphonuclear cells. Blood culture shows the growth of the infecting micro-organisms in most cases.

Subacute bacterial endocarditis.—The onset is insidious. Gradually increasing pallor, weakness and irregular low fever are common early symptoms. Most patients are sufferers of chronic rheumatic valvular disease. Characteristic signs are:—

Fever.—Only one or two degrees of rise is common. The fever is very irregular and spontaneous periods of remission occur.

Spleen.—Moderate enlargement of spleen is very constant. Rarely, it may reach a large size.

Clubbing of the fingers, severe anaemia, and a *brownish pigmentation* of the skin are present.

Heart.—Murmurs due to previous valve disease are very commonly present. Appearance of new murmurs or change of old ones may occur. Some enlargement of the heart is common. In late stages, congestive cardiac failure from prolonged toxic effect on the myocardium develops.

Embolism.—Signs of embolism in the skin or various organs are of very great diagnostic importance. In the skin, there may appear petechial spots or painful red and raised areas. In the pads of fingers and toes, such painful nodes may develop and are called Osler's nodes. Embolism of the cerebral vessels with hemiplegia, monoplegia etc., or of the retinal vessels with blindness may occur. Renal embolism may occur with frank or microscopic hæmaturia. Splenic infarcts cause pain and tenderness and friction rub over the organ.

Purpura.—Apart from embolism, purpuric spots may appear in the skin, conjunctiva or mucous membrane due to increased fragility of the capillaries.

Renal involvement.—Albuminuria with pus cells, red blood cells and casts are commonly present. Rarely, renal œdema due to diffuse glomerulonephritis may occur and sometimes uræmia may set in.

Blood changes.—Leucocytosis is not constant and a mononuclear increase may occur. Blood culture shows growth of Streptococcus viridans in most cases.

Diagnosis.—Sometimes diagnosis is made only post mortem specially in acute cases. Evidence of multiple arterial embolism is most important in diagnosis. Presence of endocarditis, clubbing of fingers, enlarged spleen, and positive blood culture are also of help in subacute cases.

Course and prognosis.—Both the varieties are almost always fatal. In acute type, the course is very short, death occurring in six weeks. Subacute cases run a variable course from 6 months to 3 years. Death is due to chronic toxæmia and progressive exhaustion, cardiac failure, embolism of the coronaries or cerebral vessels, secondary infections or uræmia.

Treatment.—*Prophylactic.*—In those who are predisposed, chances of infection should be reduced to minimum. Septic foci should be attended to.

Curative.—Very little can be done in the way of treatment. All general measures for improvement of general health and resistance should be adopted.

For the control of infection, all attempts with the so-called blood-antiseptics have failed.

Chemotherapy with sulphanilamide group of drugs has not also met with any appreciable success specially in the subacute cases.

Recently, treatment with penicillin is said to have been beneficial in some cases.

Heparin can be given during the course of chemotherapy to increase the chances of destruction of the micro-organisms and prevent formation of fresh vegetations.

CHAPTER XI

NEUROCIRCULATORY ASTHENIA

EFFORT SYNDROME, SOLDIER'S HEART, DISORDERED ACTION OF THE HEART, DACOSTA'S SYNDROME

When symptoms, such as breathlessness, palpitation, præcordial distress, lassitude, exhaustion, tachycardia etc., which normally occur in healthy individuals after severe exertion occur frequently with slight exertion or excitement without any definite evidence of organic heart disease to account for them, the condition has been called *effort syndrome*. The term *neurocirculatory asthenia* has been suggested as a better name for the condition, because the symptoms may be excited without effort, and symptoms other than those seen normally after effort may be present.

Aetiology. *Age.*—It may occur at any age, but most commonly in young men and women.

Constitution.—A neuropathic constitution is the most important predisposing cause. The individuals are usually of nervous type, thinly built and of sedentary habits. Nervous instability is shown by their tendency to excessive sweating of palms and soles and a low symptom threshold (exaggerated symptoms on mild disturbances).

Psychological factors.—A sudden change in the mode of life, mental stress, lack of adjustment etc., (as usually occur during war conditions when a large number of people are forced into unusual hardships and danger from a comparatively secure and safe civil life), precipitate the symptoms, in those who are constitutionally predisposed.

Physical factors.—In a small number of the patients definite evidence of chronic infection like pulmonary tuberculosis or other debilitating conditions may be present. The symptoms may temporarily appear during convalescence from acute infectious fevers. In the majority however, no definite evidence of any organic physical cause can be found. The heart itself does not show any evidence of pathological change by any method of examination. It may however complicate organic heart disease when the symptoms will be out of proportion to the degree of clinical or pathological change.

Pathogenesis.—The mode of production of symptoms is obscure. An overexcitability of the nervous mechanism of stimulation of cardiac action by mild provocations and a hypersensitiveness of the sensory nerves (so that mild symptoms are exaggerated), seem to be the underlying causes.

Symptoms and signs.—The symptoms are dyspnœa, palpitation aching or stabbing pain over the mammary or submammary region rarely radiating to the left arm or angle of left scapula, lassitude and exhaustion.

The symptoms are usually excited by exertion, the degree of which varies considerably, or by excitement, heavy meals, flatulence,

anxiety, worries and psychological stress. No obvious exciting cause may be present. All symptoms may not be present in the same case. Anyone of these may be most prominent in a particular case.

The main sign is rapid pulse usually with a high pulse pressure but low diastolic pressure.

Heart.—Tachycardia, accentuated apex beat which is normally situated but widely visible or palpable. First sound over the apex is sharp and loud. Evidences of organic valvular disease are usually absent. Tenderness over the præcordium may be present.

Blood pressure. - This is usually normal. The systolic pressure may be slightly raised.

Signs of cardiac failure such as pulmonary congestion, engorged neck veins, enlarged and tender liver, œdema etc., are absent.

Physical examination of the rest of the body may discover evidence of pulmonary tuberculosis or some other chronic debilitating disease, anæmia etc.

Differential diagnosis. The importance of the condition lies in the difficulty, which sometimes occurs in distinguishing it from cardiac failure or angina pectoris of effort. This is more so because it may complicate organic heart disease. Absence of any cardiac enlargement or any sign of congestive failure, either left or right ventricular, and absence of any valvular disease, hypertension, thyrotoxicosis, etc., after careful examination to account for the cardiac symptoms would distinguish cardiac failure. In cases, where neurocirculatory asthenia complicates organic heart disease, the disproportion between the symptoms and signs, and nervous instability of the patient should lead to a correct diagnosis. The difference between anginal pain and neurocirculatory asthenia has been already described elsewhere (see page 9).

Prognosis.—Neurocirculatory asthenia is a purely functional disturbance and so does not shorten life, but it may cause severe incapacity. The patient may be incapable of any useful work because of his symptoms. With careful management however, a large number can be restored to normal function. When a definite infection like tuberculosis is found, prognosis will depend on such infections. The mental attitude of the patient and his psychological reactions to the surroundings are important factors in deciding prognosis.

Treatment.—A general reassurance of the patient and encouragement will considerably help in the treatment. If some obvious physical cause is found this should be treated. Patient should be encouraged to take exercise in open air. The amount of exercise should not cause discomfort or fatigue. Breathing exercises are also beneficial. In patients with severe symptoms, sedatives may be necessary at the initial stages. Diversional or occupational therapy may also be employed.

PART III

DISEASES OF THE URINARY SYSTEM

CHAPTER I

INTRODUCTION, INTERROGATION AND CASE-TAKING

General considerations.— The kidneys can be described as filters with a power of discretion. In the first instance, they filter out water and non-colloidal constituents of plasma and then reabsorb water and certain substances which are necessary for the maintenance of an optimum composition of the plasma and the body fluids. Thus, the waste products of metabolism or other undesirable elements are eliminated from the body without undue loss of fluids and other substances of value.

The *structure of the kidneys* consists of a collection of units called *nephrons*. Each nephron consists of three parts:-

1. *Glomerulus.*—This consists of a capillary tuft fed by an afferent arteriole from the interlobular branches of the arterial arches formed by branches of the renal artery between the cortex and the medulla of the kidney. An efferent vessel emerges from the capillary tuft and breaks up again into a network of capillaries around the renal tubules and finally drains into the veins.

2. *Bowman's capsule.*—This consists of a sac lined by a single layer of flattened epithelium, into which the glomerular tuft invaginates leaving a crescentic space between the two layers of the capsule.

3. *Tubule.*—This arises from the Bowman's capsule and consists of first convoluted tubule, descending and ascending loops of Henle, second convoluted tubule and the collecting tubules which open by ducts on the renal pyramids. The tubule is lined mostly by cubical cells except in the descending loop of Henle where the cells are flattened.

The glomeruli provide the filtration surface, the filtrate (plasma minus its colloids) collects in the Bowman's capsule and undergoes differential absorption and concentration in its passage through the tubules. It is also believed that the tubular cells may excrete some waste products from the plasma to some extent (as urea and sulphate) and form substances like hippuric acid and ammonia.

In health, all the glomeruli in the kidneys (about 2,000,000) do not function simultaneously. A large number are in a state of collapse and are held as a reserve for times of increased work.

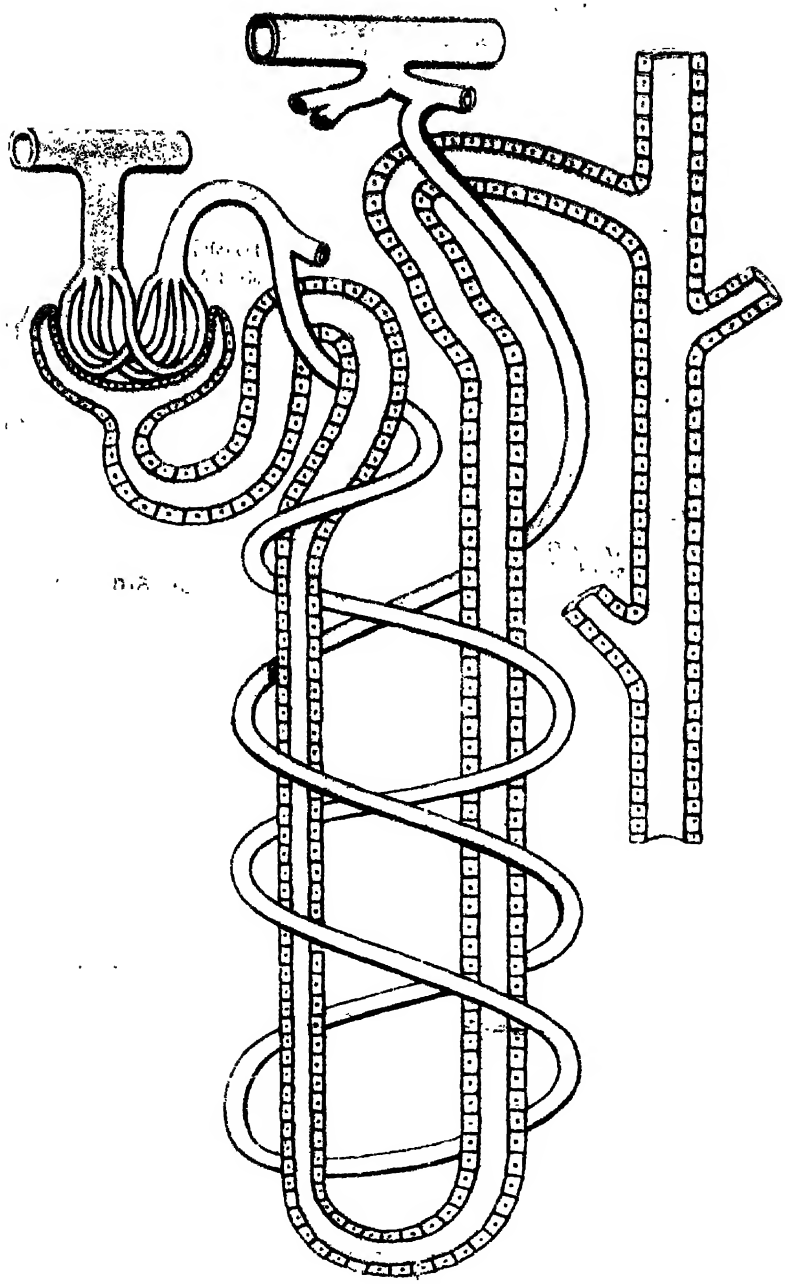


Fig. 30. Structure of a nephr

The function of the glomeruli is more or less passive, namely filtration. The amount of filtration is controlled by the number of glomeruli functioning at a given time and therefore depends on the amount of blood circulating through the kidney. It also depends on the arteriolar pressure. Glomerular filtrate may vary from 40 to 60 litres a day. The total urinary output however is much smaller (about one and half litre) indicating the remarkable power of the tubules for absorbing water. Tubules also absorb certain substances from the glomerular filtrate like glucose and chloride provided they are present in amounts less than a certain maximum. These are therefore called threshold substances. Others like urea, uric acid, etc., are not absorbed at all (non-threshold substances). These therefore undergo enormous concentration in urine. This concentrating power of the kidney is the most important indication of its functional efficiency and is measured by the specific gravity of the urine. The lowering of concentrating power and fixation of the specific gravity at a low constant level irrespective of fluid intake are indications of severe impairment of kidney function.

Manifestations of kidney diseases.—The first effect of pathological changes in the renal units is an alteration in the quality and quantity of the urine. Of these changes the presence of albumin in the urine and of some formed elements like casts in the urinary deposits is most significant. The former indicating increased permeability of the glomerular capillaries and the latter, degenerative changes in the tubules. Another important change is in the specific gravity of urine; a low specific gravity when fluid intake is restricted being an indication of deficient kidney function. The significance of these abnormalities will be more fully discussed later on.

When pathological changes in the kidneys are severe enough to impair the function sufficiently, they fail to act as regulators of blood and body fluids and a complex disturbance of metabolism develops known as *uraemia*. The earliest manifestation of such disturbance is shown by an abnormal accumulation of urea and other nitrogenous waste products in blood (*azotaemia*).

Besides these, two other important manifestations associated with kidney disease are *oedema* and *hypertension*. Although their connection with certain forms of kidney diseases has been established, their pathogenesis is still a matter of discussion.

Classification of kidney diseases. Since the time of Richard Bright who for the first time gave a comprehensive description of a group of cases and correlated them with pathological changes in kidneys, it has become customary to describe all non-suppurative and non-neoplastic diseases of the kidneys as Bright's disease. With the advancement of our knowledge it was found that under the general description of kidney diseases by Bright there were several different entities with very different ætiology and pathogenesis. A

period of confusion in terminology and in correlation between clinical findings with post mortem changes then followed.

It is now customary to classify Bright's disease on a pathological basis into three main groups--*inflammatory, degenerative* and *vascular*. The site of primary involvement is also different in the three groups. Thus, the inflammatory group first starts in glomerulus, the degenerative in the tubules and the vascular diseases in the arterioles supplying the nephrons. But as these structures are interdependent on each other, wherever the disease starts others may ultimately be affected. A mixed picture may thus be produced which was the cause of so much confusion before.

Thus the diffuse glomerulonephritis of the first group starting as an inflammation of the glomeruli in the acute stage leads to extensive fatty degeneration of the tubules in the subacute stage and atrophy of the nephrons with replacement fibrosis and vascular sclerosis in the chronic stage. It may therefore resemble lipoid nephrosis of the degenerative group in the subacute stage and the arteriolosclerotic kidney of the vascular group in its chronic stage both pathologically and clinically.

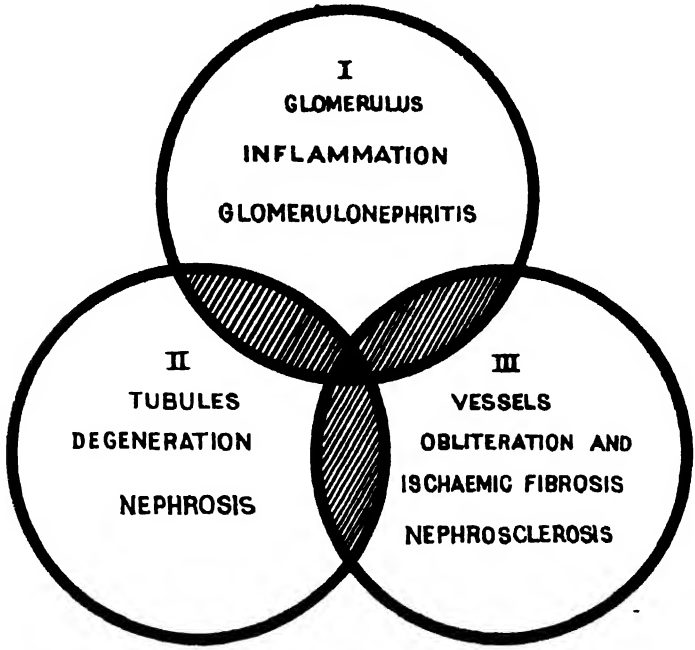


Fig. 31. Three overlapping circles showing the inter-relation between the three types of kidney diseases.

These facts have been illustrated by three overlapping circles representing the glomeruli, tubules and the blood vessels with interstitial tissue. (Fig. 31).

CLASSIFICATION OF BRIGHT'S DISEASE

I. Inflammatory diseases—Nephritis.

A. Glomerulonephritis.

Diffuse

Acute, subacute, chronic, latent.

Short description—Probably starts as an allergic sensitisation against a focal strepto coccal infection causing widespread capillary damage and inflammation in the glomerulus. Subsequent changes secondary to obliteration of glomerular capillaries are degeneration and atrophy of the nephrons and their replacement by fibrous tissue.

Embolic.

Due to bacterial embolism in the glomerular vessels as in subacute bacterial endocarditis.

Focal

Non-embolic.

Focal inflammation in the glomerulus in course of acute infective fevers or focal streptococcal infection.

B. Acute interstitial nephritis.

A fatal complication in some acute infective fevers like diphtheria and scarlet fever. Infiltration of interstitial tissues with lymphocytes and plasma cells. Cannot be recognised clinically during life.

II. Degenerative diseases—Nephrosis.

A. Larval nephrosis.

Cloudy swelling in tubular epithelium as in severe anæmia, prolonged jaundice, diabetes mellitus and mild toxæmias associated with febrile conditions.

B. Necrotising nephrosis.

Severe degenerative changes with necrosis of tubular epithelium as in severe toxæmias, mercurial poisoning cholera etc.

C. Lipoid nephrosis.

An obscure disease of unknown aetiology. A primary fatty degeneration of the tubular epithelium.

D. Amyloid kidney.

Secondary to prolonged suppuration in the body. There is a deposition of amyloid material in the walls of blood vessels and in fibrous tissues.

III. Vascular diseases—Nephrosclerosis.

A. Kidney of hypertension (Arteriolosclerotic kidney). Benign and Malignant.

Secondary to essential hypertension with consequent arteriolosclerotic changes in afferent glomerular arterioles.

B. Senile atheromatous kidney.

Atheroma in larger branches of renal arteries followed by ischæmic degeneration and fibrosis of widely separated areas of kidney substance.

Investigation and case-taking. Age.—The ætiology of kidney diseases is different at different ages. In childhood and adolescence, glomerulonephritis, acute or subacute, is most common. The chronic stage may run into adult age. In middle-aged patients, kidneys are mostly affected by arteriolosclerosis accompanying hypertension and in old age by atheroma. The kidney changes accompanying malignant type of hypertension occur at earlier age and such cases are often confused with chronic glomerulonephritis.

Sex.—Glomerulonephritis occurs in both sexes almost equally. In females, pregnancy is an important cause of nephrosis, and manifestation of latent or occult nephritis during a pregnancy should also be remembered.

Occupation.—This is unimportant so far as glomerulonephritis is concerned. In cases of hypertensive kidneys the same consideration applies as in hypertension.

Complaints.—A. *Urinary symptoms.*—(*i*) *Increased frequency* of micturition may be due to actual increase in the total quantity of urine which is called *polyuria;* or it may be due to increased irritability of the bladder or urinary passages so that a constant desire for micturition is present. This may be due to pyelitis, cystitis, urinary calculi, abnormal constituents in the urine, nephritis, tuberculosis of kidneys etc., or reflex irritation from neighbouring organs. Causes of polyuria will be discussed later.

Nocturnal frequency is characteristic of chronic nephritis or nephrosclerosis due to vascular causes. When the heart is failing the improved circulation during sleep due to rest to the heart causes increased urinary flow.

(*ii*) *Oliguria* (scanty urine) or *anuria* (absence of urine) may be due to mechanical obstruction to urinary passages, disturbances in the nerve supply of the bladder or due to actual diminution in the secretion of urine. Causes of oliguria or anuria will be discussed later.

(*iii*) *Dysuria* or difficulty in micturition is mostly due to surgical conditions in the bladder or urethra.

(*iv*) *Haematuria* or blood in urine may vary considerably in quantity from a few microscopical red blood cells in urine to frankly hæmorrhagic urine. Small quantities of blood when intimately mixed with urine gives it a turbid smoky appearance. In cases of hæmaturia it should be noted whether the blood comes at the start or at the end of micturition or whether it is intimately mixed with urine. Blood at the start of micturition is usually from the urethra and blood at the end of micturition is from the bladder. When the blood is intimately mixed with urine it is of renal origin. Causes and differential diagnosis will be discussed later on.

B. *General symptoms.*—(*i*) *Dropsy or Œdema.*—Œdema of more or less generalised distribution occurs in acute and subacute

glomerulonephritis. Œdema first appears in eyelids and face because of the loose intercellular spaces. When œdema develops rapidly it appears almost simultaneously all over the body. The severity of œdema may vary from slight puffiness of the face specially in the morning to extensive generalised swelling or anasarca with accumulation of fluid in the serous cavities. The patient has a very characteristic appearance in such a state.

The pathogenesis of œdema, its causes, differential diagnosis and treatment will be described later on (see page 123).

(*ii*) *Cardiovascular symptoms* secondary to hypertension (see page 59) or heart failure may be present specially in chronic glomerulonephritis and occasionally in the acute stage. They may also be present in hypertensive nephrosclerosis.

(*iii*) *Nervous symptoms* of hypertensive encephalopathy (see page 59) occasionally mark the onset of acute glomerulonephritis in children. It may also occur in the chronic stage of nephritis. Other nervous symptoms are; those due to uræmia and may occur in the course of chronic and less commonly of acute glomerulonephritis, in hypertension with kidney changes or in severe cases of acute nephrosis. These symptoms are headache, drowsiness or lethargy, twitchings and convulsions, loss of consciousness etc.

(*iv*) *Gastro-intestinal symptoms.*—These also appear when uræmia develops. Symptoms are anorexia, nausea, vomiting, diarrhœa, hiccough.

(*v*) *Respiratory symptoms.*—Increased rate and depth of respiration specially with a hissing sound occurs in uræmia. Less often paroxysmal dyspnœa called *renal asthma* may occur. Very rarely œdema of glottis or lung may complicate subacute nephritis causing sudden intense dyspnœa.

(*vi*) *Visual symptoms.*—Dimness of vision due to retinal changes may occur in chronic or rarely in acute stages of glomerulonephritis and in hypertension with kidney disease.

(*vii*) *General growth and development.*—Chronic kidney disease from early childhood leads to stunting of growth or infantilism and deformity of bones from disturbance of calcium metabolism called *renal rickets.*

History of past illness.—As changes in the urine and the general symptoms simulating those of kidney diseases may be produced by a large number of extrarenal conditions, the past history should be recorded according to the general plan, but special enquiry must be made for the following:—

(*i*) Symptoms suggesting early stages of glomerulonephritis which are often too inconspicuous to attract attention, *e.g.,* slight puffiness of the face or eye-lids specially in the morning after getting up from bed, following attacks of febrile cold, tonsillitis or other acute infective fevers.

(*ii*) Conditions which are known to predispose to nephritis such as:—

(*a*) Acute infective fevers like scarlet fever, measles, diphtheria or smallpox.

(*b*) Acute or chronic infections of the upper respiratory tract and middle ear.

(*c*) Impetigo and pyodermia.

(*iii*) Hypertension. Cardiac or nervous symptoms of hypertension should be asked for specially in elderly people.

(*iv*) In patients with œdema, early symptoms of heart failure, causes of heart disease, conditions that may lead to anæmia and cachexia.

(*v*) Administration of drugs containing mercury, arsenic, gold, bismuth etc., which may cause mild or severe nephrosis.

(*vi*) Prolonged suppurative processes in the body which may lead to amyloid nephrosis.

Family history.—Except in the hypertensive kidney disease, familial incidence is not common in kidney diseases.

Personal history.—Conditions predisposing to streptococcal infection of the upper respiratory tract such as dampness, overcrowding and unhygienic surroundings are important.

PHYSICAL EXAMINATION

General examination.—*State of consciousness.*—Unusual lassitude or drowsiness or actual coma in the presence of kidney disease indicates uræmia.

State of nourishment.—Emaciation, and in children retarded growth occur in chronic nephritis. Inanition due to any cause may cause œdema and may simulate renal dropsy.

Pallor.—Severe pallor is common in acute and subacute nephritis. In chronic nephritis or in chronic uræmia also there are anæmia and pallor. Severe anæmia due to other causes may cause puffiness of face and ankles etc., simulating renal dropsy.

Œdema.—Severe generalised œdema is called *anasarca* and is characteristic of subacute nephritis, less often of acute nephritis. It is also a characteristic sign of the very rare disease lipoid nephrosis. In renal œdema the face is early and severely affected. The eyelids almost close the palpebral fissure and œdema below the chin produces a double-chin appearance.

In chronic nephritis or in kidney of hypertension œdema of dependent parts only may be present due to congestive cardiac failure from hypertension.

Dyspnoea.—In contrast to heart failure, even severely œdematous patients in subacute nephritis are free from dyspnœa. This may however occur when there is heart failure secondary to hypertension or when uræmia develops. Very rarely œdema of glottis may cause intense dyspnœa.

Purpura.—Rarely purpuric hæmorrhages may occur in the skin in uræmia.

Examination of the urinary system.—The kidneys being situated deeply in the abdomen are not easily accessible to physical methods of examination. Except in cases of tumours, hydronephrosis or polycystic kidneys, physical examination of the kidneys is not of much clinical importance. The upper pole of the right kidney reaches upto the lower border of the eleventh rib. The lower pole of the right kidney is one inch and that of the left one and half inch above the highest point of the iliac crest and they lie about three inches away on either side of the middle line.

Fig. 32. Method of palpation of the right kidney.

Palpation of the kidneys.—To palpate the right kidney, the physician stands on the right side of the patient who is lying on his back with the knees drawn up. The left hand is placed at the back of the patient just below the last rib and outside the quadratus lumborum muscle and pressed forwards. The right hand is placed on the abdomen with the fingers pointing to the middle line about one inch above the umbelicus and outside the lateral border of the rectus. The hands are pressed so as to approach each other and the patient is asked to take a deep breath. The lower pole of the kidney will be felt by the upper border of the fingers at the end of inspiration specially in patients with lax abdominal wall.

The left kidney can be similarly felt standing on the left side and using opposite hands as before.

In movable kidney, owing to the looseness of the supporting ligaments, the kidneys are abnormally mobile with respiration and even the upper pole may be palpable. When a kidney can be moved about in the abdomen it is called *floating kidney.*

Swellings of the kidneys, either neoplastic, hydronephrotic or polycystic, project forwards owing to less resistance and are readily felt by abdominal palpation. There is a characteristic band of resonance over renal swellings due to the position of the colon in front.

Tenderness on palpation of the kidneys or on pressure in the renal angle behind may be due to stone in the kidney, pyelitis, pyelonephritis, tuberculous kidney or perinephric inflammation.

Examination of urine.—Diseases of the kidneys are always associated with some alteration in the urine. Therefore, this is the most important investigation in cases of suspected kidney disease. The details of the examination of urine and the significance of the urinary abnormalities will be described under special investigations.

Examination of the cardiovascular system.—This should be done according to the routine procedure for the examination of the heart and blood vessels. Special attention should be given to blood pressure, condition of the arteries, left ventricular hypertrophy and signs of cardiac failure.

Some rise in blood pressure is common in glomerulonephritis and is the rule in its chronic stage. Evidences of left ventricular enlargement, vascular changes and cardiac failure depend on the duration and degree of hypertension.

Raised blood pressure with its accompanying cardiovascular changes due to essential hypertension may also be associated with urinary abnormality or inefficient kidney function.

Examination of the respiratory system.—This should be done according to the usual routine procedure.

Wheezing and rales with paroxysmal dyspnœa resembling asthma may occur in uræmia.

Occasionally, secondary infection with bronchitis, bronchopneumonia, pleurisy etc., may complicate subacute or chronic nephritis.

Œdema of the lungs or glottis is a very rare complication in acute or subacute nephritis.

Examination of the alimentary system.—Gastro-intestinal symptoms may be very prominent in cases of uræmia. These have been already mentioned.

In the mouth and oral cavity, a heavily furred dry tongue, stomatitis, bleeding gums and foul odour are important signs of uræmia.

In the abdomen, very little can be found by examination. Ascites associated with general anasarca occurs in subacute nephritis and lipoid nephrosis.

Examination of the nervous system.—Patient's mental condition and state of consciousness should be noted. Drowsiness, lethargy, stupor or coma are signs of uræmia.

Objective signs of nerve lesion are not common except as temporary phenomena in hypertensive encephalopathy accompanying acute or chronic nephritis. In uræmic coma, the deep reflexes may be lost and plantar reflexes may be extensor in character.

Examination of the retina.—The fundus oculi should be examined with an ophthalmoscope after dilating the pupil with a few drops of lotio homatropine ½%. The optic disc should be examined for the presence of any œdema, the retina for any white exudates and hæmorrhages, and the blood vessels for any arteriosclerotic changes.

Changes in the retina like those described under hypertensive neuroretinopathy (see page 32) are also seen in chronic nephritis and less often in acute nephritis. These changes are equally ominous as in cases of essential hypertension. The exact pathogenesis of these changes is still uncertain but their association with hypertension and vascular changes has been established.

Special Investigations.—I. Examination of the urine. A. *Physical characters.*—1. *Quantity in twenty-four hours.*—Normally, about two-thirds of the total fluid intake is excreted as urine in twenty-four hours. An increase in the total secretion is called *polyuria,* a decrease is called *oliguria* and complete absence of renal secretion is called *anuria.*

Causes of polyuria.—(*a*) Excessive intake of fluid. (*b*) Diabetes mellitus. (*c*) Chronic nephritis and nephrosclerosis (due to loss of concentrating power, the kidneys try to compensate by producing large volume of dilute urine). (*d*) Diabetes insipidus (due to deficiency of posterior pituitary). (*e*) Administration of diuretics like alkaline citrates and acetates, urea, caffeine derivatives, mercurial preparations or taking of tea and coffee. (*f*) Nervous excitement.

Causes of oliguria.—(*a*) Diminished intake of fluid. (*b*) Excessive loss of fluids from the body by purging, vomiting and sweating. (*c*) States of circulatory collapse as shock and hæmorrhage. (*d*) Passive venous congestion of the kidneys as in congestive cardiac failure. (*e*) Acute and subacute nephritis and acute nephrosis. (*f*) In hæmoglobinuria due to blackwater fever, unmatched transfusion etc. (*g*) Intestinal obstruction and paralytic ileus. (*h*) Crush injuries.

Causes of anuria (*suppression of urine*).—There is no secretion of urine and the bladder is empty.

(*a*) Conditions causing oliguria may in extreme cases proceed to anuria.

(*b*) Reflex anuria—operations on kidneys or bladder may cause reflex suppression of urine.

Retention of urine.—In such cases, the bladder is distended and the patient cannot pass urine in spite of strong attempts due to (*a*) mechanical obstruction in the urethra such as by stone, stricture or by enlarged prostate, (*b*) disturbance of the nerve supply to the bladder so that the reflex co-ordination between the sphincter and detrusor muscles is lost as in spinal injuries.

2. *Colour of the urine.*—Normally the colour of the urine is light straw yellow. The depth of the colour depends on concentration of the pigments. The normal urinary pigments are urochrome and urobilinogen, the latter turns into urobilin on standing.

Dark coloured or high coloured urine may be due to—

(*a*) *Increased concentration*—In conditions of oliguria where the kidney function is unimpaired, urine of high specific gravity is produced and pigments are concentrated to produce a deep colour.

(*b*) Excess of urobilinogen and urobilin as in some (i) febrile states such as malaria, influenza, (ii) hæmolytic jaundice and (iii) defective liver function.

(*c*) *Presence of bilirubin* as in obstructive jaundice.

(*d*) *Presence of blood* either as hæmaturia or hæmoglobinuria.

The various changes in colour of the urine with their causes are shown in table vi.

TABLE VI.

Colour.	Cause.	Identification.
Deep orange, yellow.	Concentrated urine or excess of urobilinogen and urobilin.	Excess of urobilinogen gives cherry red colour with Ehrlich's diazo reagent. Urobilin produces a characteristic spectrum on spectroscopy.
Reddish orange, reddish brown or cherry red.	Administration of Prontosil rubrum, Pyridium, Amidopyrin, Aniline dyes with sweets, Beetroot, rhubarb or senna.	
Dull red, red or dark reddish brown.	(a) Presence of blood (haematuria) or altered blood (methaemoglobinuria). (b) Administration of rhubarb or senna.	(a) Red blood cells on microscopy or chemical tests for blood and spectroscopy. (b) Turns pink with alkalies.
Port-wine colour.	Haematoporphyrin in urine due either to congenital metabolic error, sulphonal or sulphanilamide poisoning or cirrhosis of liver.	Characteristic spectrum on spectroscopy.

TABLE VI.—*Contd.*

Colour.	Cause.	Identification.
Brownish-black.	(a) Excess of haemoglobin or methaemoglobin as in blackwater fever.	(a) Characteristic spectrum, on spectroscopy.
	(b) Alkaptonuria due to presence of homogentisic acid derived from incomplete breaking down of tyrosine (a congenital metabolic error).	(b) Urine turns black on standing or addition of alkalies. Reduces Fehling's solution but does not ferment with yeast.
	(c) Melaninuria in malignant melanoma.	(c) Black precipitate with ferric chloride solution dissolved in excess of the reagent giving black solution.
	(d) Excess of bile.	(d) Gmelin's test with nitric acid or other tests for bilirubin.
Deep Yellow.	(a) Administration santonin. (b) Small quantities of bile.	(a) Turns rosepink with alkalies. (b) Gmelin's test or other tests for bilirubin.
Greenish or greenish black	Administration of carbolic acid, salol, creosote, naphthol etc.	White or yellow precipitate with bromine water. There may be reduction with Fehling's or Benedict's solution.
Blue.	Administration of methylene blue.	Colour disappears on boiling urine with glucose after making it strongly alkaline with NaOH.

3. *Transparency of the urine.*—Normally, the urine should be clear and transparent. Cloudiness and turbidity may sometimes occur.

The common causes of turbid urine are—(*a*) excess of phosphates in alkaline urine, (*b*) pus in the urine and (*c*) blood in the urine.

Rarely excess of urates, oxalates or mucus may give rise to cloudiness. A milky white appearance occurs in chyluria due to rupture of obstructed lymphatics in filariasis.

Microscopical examination of the centrifuged deposit readily distinguishes the crystals of phosphates, oxalates or urates. Pus cells and red blood cells can also be easily identified.

A fine opalescence persisting after filtration is due to presence of bacteria in the urine.

4. *Reaction of urine.*—This may be either acid or alkaline depending on various extrarenal and metabolic factors.

5. *Specific gravity of urine.*—The specific gravity of the urine depends on the concentration of its dissolved constituents. The kidneys possess wide power of concentrating the urine according to the fluid needs of the body. On a restricted fluid intake, the healthy kidneys can produce a highly concentrated urine of small volume but of high specific gravity (1025 or above), on the other hand a large draught of water will produce a large volume of dilute urine with very low specific gravity.

As the concentrating power of the kidneys depends on the vital activity of the renal tubules, the specific gravity under restricted conditions of fluid intake is a reliable index of the functional efficiency of the kidneys. When renal function is impaired a large volume of dilute urine is produced to keep the total excretion of solids in twenty-four hours constant as a compensatory measure, and even if the fluid intake is restricted a high specific gravity of urine cannot be reached.

It has been seen that a specific gravity of about 1010 is the limit to which the concentrating power of the kidneys can be lowered by progressive damage. The power of producing dilute urine of low specific gravity is also gradually lost in progressive damage to the kidneys, so that finally the specific gravity of the urine remains constant near about at 1010 independent of the fluid intake. This is called *fixation of specific gravity* and is an indication of severe impairment of kidney function.

Specific gravity of a random sample of urine is of no significance so far as renal function is concerned. This should be measured under standard conditions of fluid intake. For the concentration test, the patient should be on a fluid free diet for twenty-four hours and the specific gravity of urine collected during the second twelve hours of this period should be measured. This should be over 1025, frequently it reaches 1034 or over in healthy individuals.

Specific gravity is also high when there is sugar in the urine as in diabetes mellitus.

B. *Abnormal chemical constituents.*—Albumin, sugar, ketone bodies, bile pigments and salts and indican are the common chemical abnormalities present in urine under pathological conditions.

Except albumin, the presence of the other abnormal constituents in urine does not depend on the pathological conditions of the kidneys, but on extrarenal or metabolic factors. The kidneys only excrete them as toxic waste products when produced in the system (as ketone bodies, indican) or when their concentration in blood rises above the renal threshold (as glucose, bilirubin).

Albumin in the urine should be looked for in all cases suspected of kidney disease.

Causes of albuminuria.—The term albuminuria does not mean the presence of albumin alone, but it includes all proteins which may be present in the urine giving positive reaction with the common tests. It includes serum-albumin, serum-glogulin, mucin etc. Serum-albumin however is most commonly and abundantly present in urine. (See appendix B.)

(*a*) Benign, functional or orthostatic albuminuria.—In some young persons and adolescents, albumin may be present in urine without any other evidence of kidney disease or of impaired kidney function. In such cases, albumin is particularly present in urine passed after the patient has been in erect posture for some time, whereas the first urine passed after waking up from sleep in the morning may be free from albumin. In athletes, urine passed after severe muscular exertion also has been found to contain albumin.

(*b*) All varieties of Bright's disease.

(*c*) Passive venous congestion due to (i) congestive cardiac failure, (ii) pressure on renal veins by huge ascites or abdominal tumours, (iii) hypostasis in comatose patients.

(*d*) Residual albuminuria.—Sometimes after an acute attack of glomerulonephritis, kidneys may completely recover, but slight albuminuria persists.

Mechanism of albuminuria.—As has been mentioned in the introduction, the glomerular capillaries and the epithelium of the Bowman's capsule are impermeable to colloids of the plasma, the main bulk of which consists of the proteins, albumin, globulin and fibrinogen. When, however, the permeability of the capillary endothelium is increased, larger colloid molecules are allowed to pass. As albumin has the smallest molecules amongst the colloids, it is found in greatest amount in urine; globulin is only rarely found and fibrinogen least often. It is believed therefore, that albuminuria in renal diseases is due to passage of plasma albumin through hyperpermeable glomeruli.

It is also known that proteins, which are not normally present free in the plasma, if present as such are readily excreted in the urine as foreign elements. Thus, hæmoglobin, if free in the plasma as in conditions of hæmolysis is readily excreted in the kidney and this is accompanied by escape of native plasma albumin. Similarly, egg albumin or any other foreign protein injected into circulation readily passes out in urine. It has therefore, been suggested that some alteration in the composition of plasma proteins is the cause of albuminuria in certain kidney diseases as lipoid nephrosis, because no demonstrable damage to the glomerulus is found in this disease. Such change in the plasma proteins however, has not been demonstrated.

The suggestion that albumin may be added to the urine from degeneration and disintegration of the tubular cells, also lacks support. Even if some amount of protein may be thus derived, this

cannot explain the heavy albuminuria seen in severe tubular degeneration as in subacute nephritis or lipoid nephrosis.

Other proteins and proteoses in urine.—Proteins other than albumin and globulin such as Bence-Jones protein, proteoses, nucleoproteins, hæmoglobin etc., may appear in the urine. Their presence however, is not associated with kidney disease.

Bence-Jones protein, which is precipitated at 40° to 55°C and dissolved on boiling, is present in urine of patients with multiple myeloma.

Proteoses are not coagulated by heat and are precipitated on saturation with ammonium sulphate. These are present in cases of pneumonia, asthma or when there is tissue autolysis in the body.

C. *Microscopical examination of urinary deposits.*—The urinary deposits are of two groups—unorganised and organised.

Unorganised deposits.—(*a*) Phosphates, uric acid, urates and oxalates are present normally depending on the reaction and concentration of the urine, nature of food intake and some metabolic factors.

(*b*) Certain amino-acids like cystine, tyrosine, and leucine may appear in the urine in crystalline form. Cystinuria is due to an inborn error of metabolism while tyrosine and leucine crystals in urine occur in acute yellow atrophy of liver (acute hepatic necrosis).

Organised deposits.—These are more important as indicators of local pathological changes in the kidneys.

(*a*) *Red blood corpuscles.*—These may escape in the urine due to local damage or increased permeability of the glomerular capillaries or they may be added to the urine due to hæmorrhage in the urinary passages from local disease or injury in the kidney, ureter, bladder and urethra, and from general hæmorrhagic states. The causes of hæmaturia will be discussed later.

Fig. 33. Urinary casts.—(a) Hyaline cast, (b) Granular cast, (c) Epithelial cast, (d) Blood cast.

(*b*) *Pus cells.*—Presence of pus cells in urine indicate inflammation or suppuration in the kidney or urinary passages. Causes and

differential diagnosis of pyuria (pus in urine) will be discussed later on.

(*c*) *Epithelial cells.*—Various forms of epithelial cells may appear in the urine. Depending on their source, they may be spherical, cubical, polygonal, columnar or squamous in type. Epithelial cells of renal origin may appear in acute nephritis. Irritative conditions in the urinary passages may produce large number of epithelial cells in urine. The clinical importance of epithelial cells in urine is, however, very little.

(*d*) *Casts.*—These are microscopical cylindrical bodies formed by inspissation of albuminous materials inside the renal tubules and therefore taking up their shape and frequently entangling various elements such as denuded or degenerated tubular cells, red blood cells etc. There are several varieties of casts such as follows:—

(*i*) *Hyaline casts.*—These are homogeneous and transparent in appearance. They are most commonly found and are of least significance when present alone. They may be present in any variety of albuminuria and in all varieties of Bright's disease.

(*ii*) *Granular casts.*—These show a coarsely granular and opaque appearance due to presence of granular debris from degenerated tubular epithelial cells. Their presence in large numbers indicates severe degenerative changes in tubular epithelium. They are present in subacute and chronic nephritis, severe cases of nephrosis, nephrosclerosis and passive venous congestion of kidneys.

(*iii*) *Epithelial casts.*—These contain epithelial cells from renal tubules. Their presence indicates denudation of tubular epithelium and they may be precursors of granular or fatty casts. They are commonly seen in acute nephritis.

(*iv*) *Fatty casts.*—These contain numerous fat droplets due to fatty degeneration of the tubular cells. Sometimes, doubly refractile lipoid material may be present. They are seen in subacute nephritis or in lipoid nephrosis.

(*v*) *Blood casts.*—When red blood corpuscles are present in the casts, they are called blood casts. They indicate some hæmorrhage in the glomerular space, as in acute nephritis.

(*vi*) *Leucocyte casts.*—These contain leucocytes. Their presence indicates escape of leucocytes from glomeruli and they may be seen in acute nephritis.

(*vii*) *Cylindroids.*—These are hyaline flat or band like bodies much longer than casts, frequently branched or folded on themselves and tapering to a point at the ends. They are of no pathological significance.

II. **Examination of blood.**—The nitrogenous waste products of metabolism are almost entirely excreted by the kidneys. In impairment of kidney function therefore, there will be a retention of these products in the blood. Certain other alterations in the blood

chemistry also occur in various kidney diseases. Hence a study of the blood chemistry is of great importance.

The following table shows the normal concentrations of various chemical constituents of blood which are likely to undergo alteration in kidney diseases.

TABLE VII

Substance.	Concentration.
Urea	20 to 40 mgm. per 100 c.c.
Non-protein nitrogen (N.P.N.)	20 to 40 mgm. „ „
Uric acid	2 to 3.5 mgm. „ „
Creatinine	1 to 1.5 mgm. „ „
Plasma proteins	7.5 per cent.
Albumin	5.6 „ „
Globulin	1.9 „ „
Cholesterol	180 to 220 mgm. per 100 c.c.
Chlorides (Plasma)	500 mgm. „ „
Phosphates	3.5 mgm. „ „
Calcium	9 to 11 mgm. „ „

Non-protein nitrogenous bodies in blood.—These are the end products of protein metabolism and consist mainly of urea, uric acid and creatinine. These can be estimated individually or collectively as total non-protein nitrogen (N.P.N.). An increase of these bodies in blood is called *azotaemia.*

The concentration of these substances in blood depends on the balance between their production and excretion, i.e., the amount of protein food and tissue wasting on the one hand and the excretory function of the kidneys on the other hand. Blood urea is most liable to be affected by both exogenous and endogenous protein metabolism and creatinine, by endogenous metabolism alone. Uric acid in blood is liable to increase in a disturbance of purine (nucleoproteins) metabolism apart from other proteins, as in gout.

Excretion through the kidney may fall either due to lack of concentrating power of the kidney (renal failure) or to a decrease in the glomerular filtration due to extrarenal factors. Azotæmia therefore can be renal or extrarenal in origin.

Causes of high blood urea and N.P.N. (azotaemia). *A. Renal*—Due to a failure of the concentrating power of the kidneys in various forms of Bright's disease. *e.g.*—

(*i*) Glomerulonephritis—Most commonly in chronic stage, less often in acute and rarely in subacute stage.

(*ii*) Kidney of hypertension—specially in malignant hypertension.

(*iii*) Necrotising nephrosis.

(*iv*) Severe and long standing cases of amyloid kidney.

B. Extrarenal.—Due to a diminished secretion of urine from extrarenal factors, although the concentrating power of kidneys remains normal.

(i) Conditions associated with dehydration, hypochloræmia, acidosis, alkalosis, shock or renal anoxia *e.g.*—severe vomiting and purging, cholera, pyloric obstruction, high intestinal obstruction, Addison's disease, peripheral circulatory failure, various causes of alkalosis, etc.

(ii) Anuria associated with mechanical obstruction in the urinary passages.

(iii) Suppression of urine associated with hæmoglobinuria *e.g.*, in blackwater fever, snake poisoning etc., and in crush injuries (crush syndrome).

(iv) Severe disturbance of liver function (cholæmia) as in acute hepatic necrosis (acute yellow atrophy), extreme cirrhosis etc.

Plasma proteins.—These are not normally excreted in the urine. When there is heavy albuminuria over a long period the plasma albumin is severely depleted resulting in a decrease in total plasma proteins—hypoproteinæmia, and the normal ratio between albumin and globulin is altered. The colloid osmotic tension of plasma consequently falls and œdema results.

Plasma proteins are diminished in subacute nephritis and lipoid nephrosis. These may also be diminished in starvation or famine conditions when intake of nitrogenous food is too low, and also in severe anæmias due to recurrent blood-loss.

Cholesterol.—This is increased in blood in subacute nephritis and lipoid nephrosis. It is not normally excreted in the kidneys and it is not increased in renal failure with uræmia. The significance of its rise is obscure.

Chlorides.—All fluids in the body contain chlorides to maintain an osmotic pressure, isotonic with blood plasma. Whenever there is a large accumulation of fluid in the body either as œdema fluid, effusion in serous cavities or as exudates (as in pneumonia), chlorides are locked up in such accumulations. Unless sufficient chloride is given in the food, there is hypochloræmia and diminished excretion of chlorides in urine. Loss of fluid from the body through channels other than the kidneys, such as vomiting, profuse diarrhœa, excessive sweats etc., also entails loss of chlorides and electrolytes with consequent hypochloræmia. In kidney diseases also blood chlorides are low or high according to whether there is œdema and fluid retention, oliguria, vomiting etc. It also depends on chloride ingestion.

Hypochloraemia is common in:—(*a*) Intestinal obstruction. (*b*) Severe vomiting and purging, (*c*) Severe burns, (*d*) Pneumonia, (*e*) Addison's disease, (*f*) Nephritis with œdema in some cases.

Hyperchloraemia may occur in—(*a*) Some cases of nephritis, (*b*) Urinary obstruction, (*c*) Eclampsia.

Phosphates.—Retention of inorganic phosphates in blood occurs in uræmia due to failure of excretion by the kidneys.

Calcium.—Serum calcium is diminished due to phosphate retention in uræmia.

Estimation of the functional efficiency of the kidneys.—

A. *Examination of urine.—*

(i) *for abnormal constituents—*

The significance of various abnormal constituents of urine has been discussed above. None of these can be taken as evidence of renal inefficiency. Thus in benign albuminuria, or in subacute nephritis and lipoid nephrosis with heavy albuminuria and urinary casts, renal function is not impaired. Albumin or casts in the urine however serve to call for a more thorough investigation as to the functional efficiency of the kidneys.

(ii) *for concentration and total daily excretion of various waste products.*

Chemical analysis of the urine should be interpreted with due regard to food and fluid intake, and the amount of circulation through the kidneys, as these factors greatly influence the excretion and concentration of the waste products in urine.

The following table shows the average concentration and daily output of various urinary constituents.

TABLE VIII

Substance	Average concentration in urine.	Average total output in twenty-four hours.
Urea	2 per cent	30 g
Uric acid	0.02 „	0.2 to 0.34 g
Creatinine	0.075 „	0.9 to 2 g
Chloride	0.6 „	10 to 13 g
Phosphates	0.15 „	2.5 to 3.5 g
Sulphates	0.18 „	1.5 to 3 g

(*iii*) *For concentrating power of the kidneys.*

It can be easily estimated by measuring the specific gravity of urine under complete fluid restriction for 24 hours as has been mentioned before (see page 113). One of the earliest indications of impairment of renal function is the reduction of concentrating power.

B. Examination of blood—

Urea, uric acid and creatinine are normally excreted in the urine and these will be retained in blood if the excretory function of the kidneys becomes inefficient. Estimation of urea and total N.P.N. for the assessment of kidney function however, is unreliable on the following grounds.

(i) Increase of these substances in blood may occur in various extrarenal conditions (vide page 117) where the function of the kidneys for excretion and concentration is unimpaired.

(ii) Little or no retention of urea and N.P.N. may occur because of compensatory polyuria, or a low protein diet, keeping the nitrogenous waste products within the functional limits of the impaired kidneys. Azotæmia occurs only when the compensation fails or when the impairment is severe.

(iii) The height of blood urea or N.P.N. is not always proportionate to the degree of renal failure, so that a patient with 100 mgms of urea per 100 cc of blood may show all symptoms of uræmia, whereas another with 200 mgms may be symptom-free.

C. Examination of blood and urine simultaneously.—It has been shown that the rate at which the kidneys can eliminate urea from blood over a given time bears a relation with its concentration in blood and the volume of urine produced during the period. This fact has been utilised in devising a test of renal function independent of diet and fluid restriction.

Urea clearance test.—(Möller, McIntosh and Van Slyke). By this test is measured the volume of blood which is cleared of its urea content in one minute. Two formulæ have been worked out depending on the volume of urine produced per minute. When this is 2 cc or more the urea clearance is 'maximum' (Cm) and when it is less than 2 cc, the clearance is called 'standard' (Cs).

$$Cm = \frac{U}{B} \times V \qquad \text{Normal average Cm} = 75 \text{ cc}$$

$$Cs = \frac{U}{B} \times \sqrt{V} \qquad \text{Normal average Cs} = 54 \text{ cc}$$

where U = concentration of urea in urine
B = " " " in blood
V = volume of urine per minute.

Method.—The patient evacuates his bladder completely and then refrains from any food or drink for 2 hours. Urine is collected at the end of 1st and the 2nd hours seperately, completely emptying the bladder each time, and the volume of each sample is measured. The urea content of each sample is also determined. The volume of urine per minute is calculated by dividing total volume of each sample by 60. Values of U and V are thus obtained, for two one-hourly periods; a mean of the two values is taken for final calculation. A sample of blood is drawn from a vein midway between the two hourly periods and urea content is determined. Value of B is thus obtained. According to the value of V, maximum or standard clearance is calculated from the above formulæ. The result of Cm or Cs as the case may be is expressed as a percentage of the normal average; 75 cc or 54 cc being taken as 100 per cent respectively.

With this test it has been shown that urea clearance of 20 to 40% of normal in some cases of nephritis may be associated with normal

blood urea, indicating the wide reserve power of the kidneys and the unreliability of the blood urea level alone as a guide to kidney function. Uræmia does not set in if the urea clearance is over 10% and uræmia is always present if clearance value falls below 5%.

D. Elimination and concentration tests.—Measured quantities of certain substances which are known to be excreted through the kidneys are administered and their excretion in the urine is noted over a given period.

(*i*) *Water elimination test.*—After complete evacuation of the bladder the patient is given 1 pint of water to drink. The urine passed in the next four hours is collected and measured. A healthy person with normal kidney function eliminates the total quantity of water within four hours. In impaired kidney function complete elimination takes a longer time. In the presence of œdema or congestive cardiac failure however this test is useless as the water is deviated to the tissue spaces and hence not available for excretion through the kidneys.

This test associated with concentration test (increase of specific gravity of urine on deprivation of water) gives very valuable information of kidney function.

(*ii*) *Urea concentration test.*—After a light meal overnight, the patient takes 15 gms of urea dissolved in 100 cc of water in a fasting stomach in the morning, after completely evacuating the bladder. He should pass the next 3 hours preferably in bed and refrain from any food or drink. Urine is collected at 1 hourly intervals for 3 hours in separate bottles with separate labels. The concentration of urea in each sample is measured. Normally the maximum concentration reached is 2% in at least one of the samples. If the maximum concentration does not reach 1.5% in any of the samples, kidney function is definitely impaired.

The disadvantages of this test are—(*a*) in œdematous patients, urea induces a diuresis and thus lowers the concentration of urine, and (b) urea causes a feeling of nausea because of its disagreeable taste in some patients and may even be vomited out.

In children from 3 to 5 years half the dose should be given; the full dose can be given from 12 years upwards.

(iii) *Excretion pyelography.*—Some iodine containing dyes when injected intravenously are readily excreted by the kidneys. and render the urinary tract opaque to x'rays because of the concentration of the dye in urine.

Dyes commonly used are Uroselectan B, Uropac and Pyelectan. 20 cc (containing 15 gm of the dye) is slowly injected in the anticubital vein; X'ray photograph taken 20 to 30 minutes after the injection clearly shows the kidneys, renal pelvis with the calyces and the ureters. Serial pictures may be taken 10, 30 or 50 minutes after the injection for better study. If the kidney function is poor, the

dye is not sufficiently concentrated and the affected kidney is not visualised. Individual kidney function may thus be tested by this method.

Besides the functional efficiency, this test helps to determine the shape, size and position of the kidneys, shape of the renal calyces, position of the ureters etc. The test is invaluable in the diagnosis of renal tumours, hydronephrosis, tuberculosis of kidneys, calculus in the urinary tract, abnormalities of ureters etc.

(iv) *Other tests.*—Elimination test with various dyes may also be done. Known quantities are injected. The quantity eliminated in the urine over a given time is noted and compared with the normal.

(a) *Phenolsulphonphthalein test.*—6 mgms is injected intramuscularly and the urine collected over 2 hours allowing 10 minutes for absorption. About 60 to 85 per cent of the dye is excreted in 2 hours normally.

(b) *Indigo carmine test.*—0.1 gm of the dye is injected intramuscularly. The intensity of colour of the dye in urine is examined as it is excreted. By collecting urine from each ureter separately by a ureteric catheter passed through a cystoscope function of each individual kidney can be compared.

CHAPTER II

ŒDEMA.

Definition.—Œdema is a condition of abnormal accumulation of fluid in the intercellular spaces and serous cavities.

Clinical features of oedema—

The presence of œdema is shown by—

(a) Swelling of subcutaneous tissues.

(b) Stretching of superficial skin with loss of normal crevices and wrinkles. Skin looks shiny.

(c) Bony prominences disappear and normal hollows on the surface are filled up.

(d) Pitting on pressure—on persistent pressure on the part with a finger a pit appears due to displacement of the œdema fluid. The pit gradually fills up again.

This is the most important sign of œdema. It distinguishes true œdema from similar swelling of subcutaneous tissue in myxœdema or elephantiasis (so called solid œdema) where no pitting occurs. Clinical manifestations of œdema do not appear until about 5 or 6 litres of fluid have accumulated in the tissue spaces. The only evidence of this latent œdema is an increase of body weight.

Distribution.—The accumulation of fluid is maximum in places where the tissue spaces are loose as in the subcutaneous tissues, particularly around the eye-lids and in the face. The common sites where œdema appears are the eye-lids, face, under the chin, around the ankles, scrotum, over the sacrum and the dorsun of the hand. Transudation into serous cavities like the pleural cavity (hydrothorax) and peritoneal cavity (ascites) may occur. Generalised œdema with transudates in serous cavities is called *general anasarca.*

Pathogenesis.—Œdema is produced by a disturbance of the factors which normally maintain an equilibrium in the exchange of fluid, carriying metabolites and gas, between the blood in the capillaries and the extracellular fluid in the tissue spaces. These factors are the (a) capillary wall which acts as a semipermeable membrane allowing passage of water and crystalloids and retaining colloids (b) intra capillary pressure which tends to force water into the tissue spaces, and (c) colloid osmotic pressure of plasma—which tends to retain water inside the capillaries.

(a) *Capillary permeability.*—While crystalloids are free to pass through the capillary wall, the colloids (plasma proteins) are retained inside the capillaries. The osmotic pressure of the colloids antagonises the intracapillary pressure and tends to absorb water from the tissue spaces which contain little or no colloids.

If the capillary wall is damaged colloids are allowed to pass into the tissue spaces. The plasma colloid osmotic pressure is thus partially neutralised, absorption of fluid from tissue spaces is dimi-

nished and œdema results. The œdema fluid in such cases is partially rich in protein (1% or more). Such capillary damage is the cause of œdema in acute nephritis and this type of œdema is therefore called *nephritic oedema.*

Increased capillary permeability may also occur in anoxæmia associated with heart failure, in severe anæmia and in allergic or anaphylactic conditions.

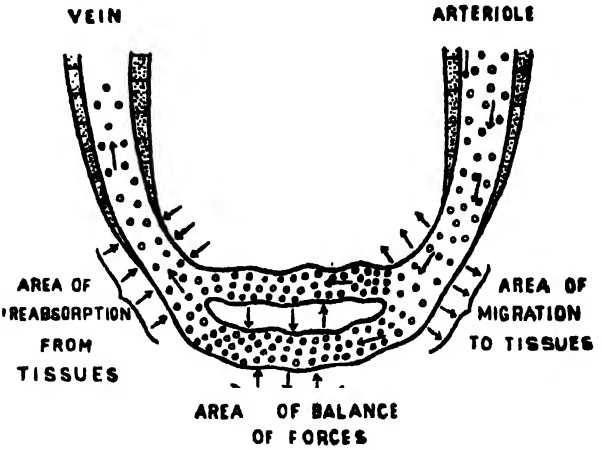


Fig. 34. Diagram showing the balance of forces controlling the normal exchange of fluids between the tissue spaces and the capillaries.

(b) *Intra-capillary pressure.*—The intra-capillary pressure falls gradually from the arterial end (about 27 mm. of mercury) to the venous end (about 13 mm. of mercury);—being higher than the colloid osmotic pressure at the arteriolar and lower at the venous end. Thus normally fluid is forced out into the tissue spaces at the arteriolar end and reabsorbed into the capillaries at the venous end. (See Fig. 34). An equilibrium exists between this migration of fluid in and out of the capillaries preventing abnormal accumulation in the tissue spaces. Increase of intra-capillary pressure will lead to œdema due to increased transudation and diminished reabsorption. (See Fig. 35).

Increase of venous pressure or venous stasis, as in congestive cardiac failure or venous obstruction, raises intra-capillary tension by back-pressure. Increase of arterial pressure, however, has no effect on intra-capillary pressure.

Rise of capillary pressure is the main factor in causing œdema in cardiac failure. This type of œdema is therefore called *cardiac oedema.* The anoxæmia from circulatory stasis in this condition increases capillary permeability which is an additional factor in

such œdema. The œdema fluid is thus partially rich in proteins (0.5%).

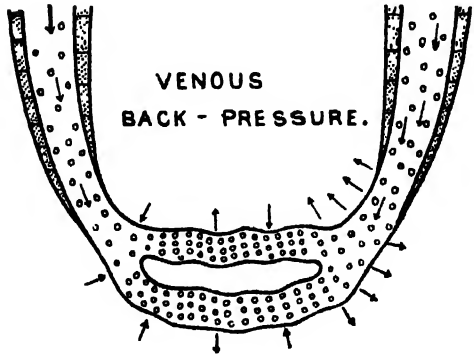


Fig. 35. Diagram showing the pathogenesis of cardiac œdema.

In the early stages the venous stasis is exaggerated in the veins of dependant parts by gravity, so that œdema first appears in the dependant parts of the body in cardiac failure.

(c) *Plasma colloid osmotic pressure.*—This is the pressure with which the colloids in the plasma tend to absorb water from the tissue spaces. The colloids of the plasma are mainly the proteins; and of the proteins albumin having the smallest molecule contributes the maximum share to the total colloid osmotic pressure. As it antagonises the capillary pressure, a reduction in the colloid osmotic pressure increases the transudation and reduces reabsorption leading to œdema. (See Fig. 36).

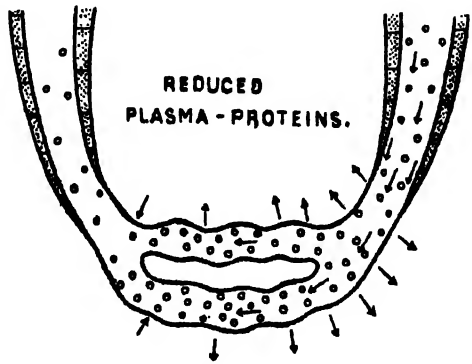


Fig. 36. Diagram showing the pathogenesis of nephrotic œdema.

A reduction of plasma proteins particularly of albumin occurs in long continued heavy albuminuria as in lipoid nephrosis or sub-

acute nephritis, specially when the diet is not rich in proteins. In these cases œdema occurs due to reduction of plasma colloid osmotic pressure and is called *nephrotic oedema.* The œdema fluid is very poor in proteins.

Hypoproteinæmia and nephrotic type of œdema may also occur in the following conditions,—(i) severe anæmia, (ii)cachexia, (iii) chronic wasting diseases, (iv)prolonged starvation, (v) advanced cases of cirrhosis of liver.

Accessory factors in the production of oedema.—These factors by themselves cannot produce œdema, but when œdema is already present due to any one of the causes already mentioned, they may further increase fluid retention in the tissue spaces.

Such factors are salt and water. It has long been known that excess of sodium chloride and water given to œdematous patients (either nephritic, nephrotic or cardiac) increase œdema and are not followed by a corresponding increase in urinary excretion of these substances. This is not due to any failure of the kidneys to excrete them but due to a prerenal deviation by which water and sodium chloride pass into the tissue spaces instead of being available for the kidneys for excretion.

That these substances by themselves would not produce œdema is shown by the fact that when there is mechanical obstruction to urinary passages leading to complete anuria for days, chlorides increase in blood enormously, still no œdema appears.

It has been shown that the sodium ion is more effective in increasing œdema than the chloride ion.

Hydrophilia of the tissues.—According to M. H. Fischer œdema is due to increased power of the tissue cells to hold water as a result of acidosis and electrolytic disturbances. This theory lacks proof and support.

Lymphatic obstruction and nervous disturbance.—Lymphatic obstruction may cause local œdema as in filariasis. Long continued lymph stasis and lymphangitis lead to a peculiar thickening of skin and subcutaneous tissues causing increase in size of the part, which is called elephantiasis.

Local œdema of short duration may occur in parts of the body due to neurovascular disturbances, as in angioneurotic œdema. It is believed to be of allergic origin.

Causes of oedema.—A. *Generalised oedema.*—

1. Kidney diseases—(a) Acute and subacute glomerulonephritis, (b) Lipoid nephrosis.
2. Congestive cardiac failure.
3. Severe anæmia.
4. Epidemic dropsy.
5. Prolonged starvation and cachectic conditions.
6. Beriberi.

(Generalised œdema may be simulated by myxœdema. Some

œdema with pitting in the legs may occur in flabby patients with poor muscular tone).

B. *Local oedema*—1. Lymphatic obstruction e.g., filariasis or malignant infiltration of lymph glands and lymph vessels. There is no pitting on pressure.

2. Venous obstruction e.g.,—(a) Thrombosis of pelvic veins causes swelling of the corresponding lower limb—*phlegmasia alba dolens*. (b) Pressure on inferior vena cava by huge ascites causes œdema of legs.

3. Angioneurotic œdema—Non-pitting.

4. Elephantiasis—commonly in the lower limbs and scrotum—Non-pitting.

5. Milroy's disease—Hereditary swelling of lower limbs. No pitting on pressure.

6. Local inflammation, bites or stings of insects, application of irritants.

7. Iodism—Œdema of eye-lids and face, after administration of iodides in people with idiosyncrasy.

Differential diagnosis.—1. *Renal oedema.* (i) Age incidence—Usually in children or adolescents. (ii) History—œdema first appears in face or simultaneously all over the body. This may be the first attack or there might have been previous attacks. No palpitation or dyspnœa on exertion preceding œdema. (iii) Onset—May be sudden or insidious with gradually increasing puffiness of the face, usually following an attack of cold, sore-throat or tonsillitis. (iv) Hæmaturia may be present at the onset in the present attack or previous attacks (except in case of lipoid nephrosis). (v) Physical signs—Characteristic puffiness of face and eye lids with severe pallor, cyanosis, engorgement of neck veins, dyspnœa or orthopnœa in spite of severe anasarca are absent. Liver is not enlarged or tender. Heart size is normal (except in cases of acute or recurrent nephritis with hypertension). (vi) Urine—Scanty, copious albumin, casts and red blood cells according to the stage or type of kidney disease. (vii) Blood chemistry—Urea and N.P.N. increased in acute nephritis. In subacute nephritis and lipoid nephrosis, cholesterol is increased and plasma proteins are diminished.

2. *Congestive cardiac failure.*—(i) Age.—May occur at any age. (ii) History—Œdema first appears in the dependant parts, as around the ankles in ambulant patients, usually preceded by breathlessness and palpitation on accustomed exertion. (iii) Past history suggestive of rheumatic infection in case of children and young people and that of hypertension or syphilis in elderly persons. (iv) Patient has dyspnœa on slight exertion or has orthopnœa. Presence of signs of congestive failure (vide page 37), Œdema—most marked in the legs, scrotum, over the sacrum in bed-ridden patients. Face not affected or slightly affected. Ascites and hydrothorax (more commonly right sided) may be present. (v)

Heart—size enlarged—signs of organic disease either valvular or myocardial are present. (vi) Liver is enlarged and tender. (vii) Urine scanty. Albumin, hyaline and granular casts and a few red blood cells may be present. Specific gravity high and excess of urates present. (viii) Blood chemistry—Plasma proteins and cholesterol within normal limits. Slight increase of blood urea however is not uncommon owing to severe oliguria, specially in cases of hypertensive heart failure where the kidneys have undergone some nephrosclerotic change.

3. *Severe anaemia.*—(i) Onset is insidious with slight puffiness of the face and ankles. Weakness, dyspnœa on exertion etc., may be present. (ii) Past history may suggest such diseases as may lead to severe secondary anæmia. (iii) Œdema is generally slight, severe pallor of the skin, conjunctiva and tongue. (iv) There is no dyspnœa or orthopnœa. Neck veins are not engorged. (v) Heart may be slightly enlarged with functional systolic murmurs. (vi) Liver is not enlarged or tender (liver and spleen may be enlarged when anæmia is secondary to diseases causing enlargement of these organs e.g., malaria, kala-azar, etc.) (vii) Urine—Quantity normal. Free from albumin, casts, red blood cells etc., (slight albuminuria may occur due to severe anæmia). (viii) Blood picture—Hæmoglobin and red blood cells greatly diminished. Characteristic picture of microcytic or macrocytic anæmia. Urea, N.P.N., cholesterol etc., normal. Low blood proteins.

4. *Epidemic dropsy.*—(i) History of epidemic œdema in the locality, or of several members of the family being affected. Affects mainly Bengalees. (ii) Mild gastro-intestinal disturbances, such as diarrhœa may be present. (iii) Œdema in the legs is commonly a solid induration of subcutaneous tissue often with a red flush; may also occur in other parts of the body. Pitting œdema may also occur in the legs. Face is not affected. There is no ascites. (iv) Angiomatous nodules in the skin, mucous membranes and conjunctivæ may be formed. (v) Visual disturbances associated with rainbow haloes around lights at night due to glaucoma may occur. (vi) Signs of cardiac failure may be present. (vii) Urine—No abnormality. (viii) Blood chemistry—Normal.

5. *Beriberi.*—(i) Occurs in people living mainly on polished rice and other diet poor in vitamin B_1. (ii) Œdema of legs with loss of knee jerks and ankle jerks, signs of heart failure may be present. (iii) Urine—No abnormality. (iv) Blood chemistry—normal.

6. *Starvation and cachectic oedema.*—(i) History of prolonged privation or chronic debilitating diseases. (ii) General emaciation and anæmia, puffiness of the face and œdema over the extremities common. No evidence of congestive cardiac failure such as engorged neck veins or enlarged or tender liver. Other signs of chronic diseases like pulmonary tuberculosis, may be present. (iii) Urine—No abnormality. (iv) Blood shows anæmia and hypoproteinæmia.

7. *Ascites due to cirrhosis of liver with oedema of the legs.*—Such cases may be distinguished by the following:—(i) A long history of chronic gastritis and flatulent dyspepsia. Ascites precedes the œdema of the legs. (ii) Face is pinched and has a characteristic earthy colour or ashen pallor. Upper limbs and chest are emaciated. Neck veins are not engorged and heart is not enlarged. Distended veins are seen over the abdomen. No dyspnœa or orthopnœa. Liver and spleen may be both felt (by dipping if necessary) to be enlarged. (iii) History of hæmatemesis may be present. (iv) Urine—scanty, contains albumin and hyaline casts. (v) Blood chemistry is normal except in very advanced stages of hepatic failure.

A similar condition of ascites and œdema of the legs may occur in constrictive pericarditis. (For details see chronic pericarditis page 91).

Treatment of oedema—1. *Restriction of fluids and sodium chloride.* As both these substances are known to increase œdema when a cause for its production is present,these should be restricted to a minimum. In the presence of severe œdema the total fluids should not exceed one pint or preferably less a day. Sodium chloride is best avoided completely in severe œdema. Ordinarily addition of common salt in the cooking and use of table salt during meals are avoided. Sometimes potassium or other chlorides are used in place of sodium chloride.

2. *Elimination of water by purgation, diuresis and diaphoresis.*

(a) *Purgation.*—Saline and hydragogue cathartics are used. Their dose should be so regulated as to avoid too much purging and prostration of the patient. One or two fluid motions a day should be aimed at.

In cases of cardiac œdema a dose of blue pill (4 to 8 grs.) at night followed by 1 oz. saturated solution of magnesium sulphate in the morning to start with and then one or two doses of mist alba early in the morning for a few days should be given.

R/-

Mag carb (levis) gr. 20
Mag sulph .. dr. 2
Aqua menth pip ad oz. 1

(*b*) *Diuretics.*—The choice of diuretics will depend on the cause of œdema and the functional condition of the kidneys. Diuretics in common use are the alkalies, urea, drugs of caffeine group and mercurial diuretics.

(i) *Alkalies.*—Acetate and citrate of potassium in doses of 15 to 20 grains are most commonly used. They are mild and non irritating and therefore can be used in the presence of even acute inflammation in the kidneys. They are most suitable in acute and subacute glomerulonephritis.

(ii) *Urea.*—In 15 gm. doses it acts as a strong diuretic but is not in common use because it provokes nausea. It can not be given where kidney function is impaired and blood urea is high.

(iii) *Caffeine derivatives.*—These are caffeine, theobromine and theophyllin. They are uncertain in their diuretic action and when patients are habituated to their use they cease to be effective. They are not suitable in the presence of active inflammation in the kidneys. They may produce gastric irritation. Those commonly used are:

(1) Theobromina et sodii salicylas (diuretin) given orally in 10 to 20 grs. doses.

(2) Theophyllina et sodii acetas—given orally in 2 to 5 grs. doses (Theocin sodium acetate).

(3) Theophyllin ethylene diamine—(euphyllin or aminophyllin) orally or intramuscularly—in 1½ to 3 grs. doses.

These drugs, specially euphyllin are very useful in cardiac œdema associated with hypertension.

(iv) *Mercurial diuretics.*—Some organic mercury preparations act as very strong diuretics when given intramuscularly or intravenously. Of these, the most commonly used drugs are *Salyrgan* (Mersalyl B.P.), *Esidrone* and *Neptal.* These drugs irritate the kidneys and may lead to serious consequences if used when the kidneys are damaged or are in a state of inflammation. All precautions, therefore, should be taken before their use to ascertain kidney function. They are most useful in non-renal œdema, such as cardiac œdema and also in serous effusions such as ascites due to portal obstruction.

Salyrgan is used in doses of 1 to 2 cc. intramuscularly or intravenously after testing for idiosyncrasy with a test dose of ½ cc. A preliminary administration of ammonium chloride gr. 20 to 30 t.d.s. for 2 days preceding salyrgan (to render the urine acid) increases the diuresis considerably. When a good response is obtained a diuresis of 100 to 200 ozs. in 24 hours may follow a single injection of 2 cc. of salyrgan. Injections may be repeated twice a week at first and then once a week if necessary.

(c) *Diaphoresis.*—Elimination of water by perspiration is not commonly practised now-a-days. Measures which produce severe perspiration, also cause severe exhaustion and prostration. Mild measures like warm bath or electric bath followed by wrapping in warm blankets from 15 to 30 minutes may be tried. But the efficacy of such methods in relieving œdema is only slight.

3. *Raising the colloid osmotic pressure of plasma when it is low.*—Hypoproteinæmia plays a main part in the production of œdema in cases of subacute nephritis, lipoid nephrosis, starvation and anæmia, as we have seen before, by lowering the colloid osmotic pressure of plasma.

In such cases the plasma proteins should be raised by a high protein diet. 120 to 220 gms. of proteins in 24 hours should be given provided the kidney function is good and blood urea is not raised.

Attempts have also been made to improve plasma osmotic pressure and induce diuresis by the intravenous injection of gum acacia solutions (6% acacia in normal saline) or concentrated human serum and protein hydrolysates. The effects of such measures are temporary.

4. *Treatment of the underlying causes of oedema.*—In congestive cardiac failure, attention should be paid to improve cardiac function and relieve venous stasis by rest, digitalisation, venesection etc., (vide page 38).

When anæmia or hypoproteinæmia are present these should be corrected by adequate hæmatinics and dietetic measures such as iron preparations, liver extract and high protein diet.

In beriberi, vitamin B_1, in large doses—25 mgm. a day, for about 3 weeks, will act as a specific.

In renal œdema, the treatment is still for the most part only symptomatic.

CHAPTER III

DIFFUSE GLOMERULONEPHRITIS

Aetiology.—1. *Age.*—Children and adolescents are predominently affected.

2. *Infection.*—A streptococcus hæmolyticus infection of the upper respiratory tract is the commonest predisposing cause. Thus acute tonsillitis, scarlet fever (fortunately rare in this country) and secondary streptococcal infection associated with common cold, influenza, measles, small pox etc., are frequently followed by acute diffuse nephritis.

Less commonly localised streptococcal infections elsewhere as in erysipelas, inpetigo, otitis media etc., may also lead to nephritis.

Infection is focal and the kidneys are not directly invaded by the organisms. Nor is nephritis toxic, as it does not occur at the height of the infection but when infection is declining (about 2-3 weeks later).

3. *Allergy.*—In the course of immunisation against the infection, a state of allergy develops and the capillaries of the body are involved in an antigen-antibody reaction. The kidney glomeruli are more severely affected because of their excretory role and therefore a greater concentration of the toxins in them.

4. *Other predisposing causes.*—Circumstances which favour streptococcal infection of the upper respiratory tract predispose to nephritis. These are dampness, cold, ill-ventilation, overcrowding, malnutrition etc.

Pathology.—In the early or acute stage there is a widespread capillary damage all over the body (capillary toxicosis), with special and more severe involvement of the glomerular capillaries of the kidneys. Over the rest of the body the damage is shown by an increased permeability of the capillaries resulting in nephritic œdema, whereas in the kidneys an inflammation of the glomerulus (*Glomerulitis*) is shown by exudation of albumin, fibrin, red blood cells and leucocytes into the capsular space and a proliferation of the lining cells of the capillaries and the Bowman's capsule. The capillaries are partially or totally blocked by proliferated cells and hyaline deposition. If the inflammation does not subside, and continues in a subacute form the second stage is reached. Both intra and extra capillary proliferation continues and the capsular space is filled up with a crescentic mass of epithelial cells (*epithelial crescent*). The capillaries are completely blocked and show signs of hyaline degeneration. The blood supply to the tubules being derived from the efferent glomerular vessel, is impaired or totally lost. Moreover, the filtration through the glomeruli being stopped from blockage of the

capillaries, the tubules have no function of absorption. The tubular cells therefore undergo degeneration. In the second stage (subacute stage) widespread fatty degeneration of the tubular cells predominate the picture and gives the kidney a typical appearance—*large white kidney*. The glomeruli with the proliferated cells gradually undergo hyaline degeneration and the rest of the nephron also undergoes atrophy from loss of blood supply and disuse. A fibrous tissue hyperplasia from the interstitial frame work of the kidney ultimately replaces the atrophied parenchyma. This is the third stage or chronic stage characterised by atrophy and replacement fibrosis. The fibrous tissue contracts diminishing the size of the kidneys. Those glomeruli and tubules which have escaped or recovered from the previous inflammation tend to undergo a compensatory hypertrophy. A granular appearance is thus produced on the surface due to healthy hypertrophied areas projecting above the contracting scarred areas.

The typical naked eye and microscopical appearances of the three stages of nephritis are described in the following table. (Table IX—Page 134). There may be various intermediate stages and both advanced and earlier changes may be found in the same kidneys. The severity and progress of the lesions are of extreme variability.

The contracted kidney of hypertension is called primary while that of chronic nephritis is called secondary. The distinguishing features between the primary contracted kindney (nephrosclerosis) and secondary contracted kidney (chronic nephritis) are the following:

(*i*) In nephrosclerosis the vascular thickening is more prominent and thickened and gaping blood vessels are seen to stick out on the cut surface of the kidneys.

(*ii*) The atrophy and fibrosis are more patchy in distribution in nephroselerosis. Normal or hypertrophied renal parenchyma is more abundant than in chronic nephritis where atrophy and fibrosis are more diffuse and widespread.

(iii) In chronic nephritis, some glomeruli still show inflammatory changes of earlier stages of nephritis, which are never seen in nephrosclerosis.

Clinical manifestations.—Correlation of signs and symptoms with the pathological changes in the kidneys in Brights' disease has always been difficult, probably because of the wide variability in the severity and the rate of progress of the lesions.

In the *acute stage,* widespread capillary damage causes generalised œdema, which may be marked, slight or none at all. Damage to glomerular capillaries results in hæmaturia and albuminuria due to escape of red blood cells and proteins, the degree of which again is variable. As some of the glomerular capillaries are blocked and there is certain amount of arteriolar spasm, total quantity of urine is diminished, in severe cases very markedly, resulting in some

TABLE IX.

Pathological changes in the Kidneys in the three stages of glomerulonephritis.

	Acute	Subacute	Chronic
A. *Macroscopical changes.*			
1. Size.	Normal or slightly enlarged.	Enlarged.	Reduced.
2. Colour.	Deep reddish brown, sometimes with minute dark spots of hæmorrhage on the surface.	Pale yellow.	Pale brown.
3. Surface.	Smooth.	Smooth.	Granular, sometimes with small retention cysts at places.
4. Capsule.	Not adherent to the underlying kidney substance. Strips easily.	Not adherent, strips easily.	Adherent to underlying kidney substance which is torn off on attempting to strip off the capsule.
5. Cut-surface.	Cortex enlarged and cloudy. Pyramids congested.	Cortex enlarged and pale. Yellow streaks of fatty degeneration are seen.	Cortex shrunken and atrophied. Cortex and medulla not well demarkated. Intrapelvic fat increased.
B. *Microscopical changes.*			
1. Glomerulus.	Swollen and more cellular due to proliferated endothelium and leucocytic infiltration.	Swollen and more cellular with evidence of hyaline degeneration in places.	Atrophied and represented by hyaline globular masses. Some glomeruli are normal and others show subacute changes.

TABLE IX.—*Contd.*

	Acute	Subacute	Chronic
2. Glomerular capillaries.	Dilated and almost filled with proliferated endothelial cells. Deposits of hyaline substance at places occluding the lumen. Most capillaries are devoid of blood.	Filled with proliferated endothelial cells. Hyaline degeneration starting in places.	Completely hyalinised in the affected glomeruli.
3. Bowman's capsule.	Slight proliferation in the visceral layer. Capsular space filled with albuminous exudate, red blood cells and leucocytes.	Epithelial crescents in capsular space. Adhesions between two layers of the capsule at places.	The proliferated cells have undergone hyaline degeneration and fibrosis. A few epithelial crescents may still be present here and there.
4. Tubules.	Lining cells show cloudy swelling. Lumen filled with hyaline casts or desquamated cells.	Widespread fatty and granular degeneration of lining cells. Lumen filled with granular debris and fatty casts.	Tubules of affected nephrons have atrophied and are replaced by fibrous tissue. Hypertrophy of the remaining tubules shown by dilatation and flattening of the lining cells.
5. Interstitial tissue.	Not affected.	Not affected.	Marked increase of fibrous tissue and round cell infiltration specially around the atrophied glomeruli.
6. Renal blood vessels.	Normal.	Normal.	Diffuse hyperplastic sclerosis and endarteritis obliterans.

retention of nitrogenous waste products in blood. Hypertension is present in almost one-third of the cases and is believed to be a compensatory measure to force blood through blocked or partially blocked glomerular capillaries. Sometimes hypertension plays an important part in the symptomatology by producing sudden left ventricular failure or hypertensive encephalopathy. Sometimes, pathological changes are so mild as to cause no clinical manifestations except urinary changes.

In the *subacute stage,* nephrotic type of œdema from prolonged albuminuria is the most obvious manifestation. The urine contains copious albumin and various casts due to degenerative changes in the tubules. The glomerular changes are as a rule very slight and so hæmaturia is not common. Renal function is generally good. Blood shows normal urea and N.P.N. but the total plasma proteins are diminished with a reversal of the normal albumin: globulin ratio because of severe albuminuria. Blood cholesterol is raised due to unexplained cause. Just as the pathological picture resembles lipoid nephrosis, so the clinical picture is also entirely nephrotic. When however the glomerular changes are more active or when acute exacerbations occur, hæmaturia, hypertension, nitrogen retention etc., are liable to appear. Sometimes this stage also may remain clinically latent.

In the *chronic stage,* as more and more nephrons degenerate and atrophy, kidney function becomes impaired. Concentrating power is also reduced. Finally hyalinisation of the affected glomeruli reduces albuminuria; casts also diminish in number. A compensatory polyuria occurs draining out the œdema. Blood pressure is raised. For a variable time the patient may remain in a state of compensated renal function but sooner or later renal failure and uræmia supervene. Vascular and cardiac changes associated with hypertension are present and hypertensive neuro-retinopathy is very characteristic.

The details of the clinical manifestations of the three stages of nephritis and of lipoid nephrosis are shown in the accompanying table. (Table X, page 137).

Course.—Bright's disease may take one of the following courses.

(*a*) The three stages may develop successively with or without latent periods in between.

(*b*) Complete recovery after the first stage.

(*c*) First two stages may remain latent and manifestations of the chronic stage may appear with hypertension and renal failure.

(*d*) Death during first or the second stage from secondary infection or uræmia.

Duration of the disease is extremely variable. Some cases end fatally within a few months to a year. Others live for years. Periods of long latency, when nothing but albuminuria, is found, are quite common.

TABLE X.

Signs and symptoms of the three stages of glomerulonephritis and of lipoid nephrosis compared.

	Acute glomerulonephritis.	Subacute glomerulonephritis.	Chronic glomerulonephritis.	Lipoid nephrosis.
1. Modes of onset.	(a) With prominent urinary symptoms—hæmaturia (frank hæmaturia in 10% of cases, microscopical hæmaturia in the rest), oliguria, dysuria, frequency of urine and lumbar pain.	(a) Insidious onset with gradually increasing œdema first appearing in the eyelids and face, weakness, pallor and scanty urine.	(a) With hypertension and its presenting symptoms (see page 59).	Insidious onset with weakness, pallor and gradually increasing œdema first appearing in the face. Scanty urine.
	(b) With constitutional symptoms—fever, chill, headache, backache, vomiting etc.	(b) Signs and symptoms continuing from the acute stage.	(b) With uræmic symptoms — headache-, anorexia-, nausea-, vomiting-, mental and muscular asthenia, muscular twitchings, diarrhœa-, dyspnœa etc.	
	(c) With acute gastro-intestinal symptoms — vomiting, diarrhœa and dysentry.			
	(d) With cardiac symptoms — those of acute left ventricular failure due to sudden temporary hypertension.			

TABLE X.—*Contd.*

	Acute glomerulonephritis.	Subacute glomerulonephritis.	Chronic glomerulonephritis.	Lipoid nephosis.
	(e) Witr cerebral symptoms of hypertensive encephalopathy — headache, convulsions, transient paralysis, blurring of vision etc.			
	(f) With severe œdema —appearing first in the face and eyelids in half the cases and around the ankles in other cases.			
	(g) With weakness, general ill health, anæmia, anorexia, with or without slight puffiness of the face specially in the morning.			
2. History	Symptoms generally follow tonsillitis or sore throat, or one of the infections mentioned in the ætiology.	A history suggesting acute nephritis with hæmaturia, œdema, oliguria etc., may be present. Often no such history is available.	Previous history suggesting an acute nephritis and a long period of œdema may be present. Rarely no such history is present.	No history suggesting previous acute nephritis.

TABLE X.—*Contd.*

	Acute glomerulonephritis.	Subacute glomerulonephritis.	Chronic glomerulonephritis.	Lipoid nephrosis.
3. General appearance.	Variable œdema. Face pale and puffy. Skin dry.	Bloated appearance. Waxy pallor, marked anasarca.	Emaciated and anæmic. Eyes bright and watery. Skin dry. Dyspnœa may be present. Stunted growth and bone deformities in children (renal rickets).	Same as in subacute nephritis.
4. Oedema.	Present in most of the cases. Nephritic in type. Severity variable.	General anasarca with ascites and hydrothorax. Nephrotic type of œdema.	Absent. When heart failure due to associated hypertension is present, cardiac type of œdema may occur.	Same as in subacute nephritis.
5. Blood pressure.	Raised in one third of the cases.	Usually normal. May show transient rise.	Raised.	Always normal.
6. Heart.	Left ventricular enlargement in cases with hypertension.	Usually normal.	Left ventricular hypertrophy and failure.	Normal.
7. Retina.	Albuminuric neuro-retinopathy in rare cases.	Normal.	Albuminuric neuro-retinopathy is commonly found.	Normal.
8. Urinary changes.				
(a) Quantity.	**Marked oliguria.**	**Oliguria.**	**Polyuria.**	**Oliguria.**

TABLE X.—*Contd.*

	Acute glomerulonephritis.	Subacute glomerulo-nephritis.	Chronic glomeruloneph ritis.	Lipoid nephrosis.
(b) Specific gravity.	High (1025 or higher).	High.	Low.	High.
(c) Albumin.	Present in copious amount.	Present in copious amount.	Slightly present.	Copious amount.
(d) Cells.	Red blood cells, leucocytes, renal epithelial cells.	Occassional red blood cells.	Occasional red blood cells.	No red blood cells.
(e) Casts.	Plenty of hyaline, epithelial and blood casts.	Moderate numbers of hyaline, granular and fatty casts.	A few hyaline and granular casts.	Moderate number of hyaline, granular and fatty casts.
9. *Blood chemistry.*				
(a) Urea & N.P.N.	Moderately raised.	Usually normal.	Increased (usually 100 mgm per 100 cc.).	Normal.
(b) Cholesterol.	Normal.	Increased.	Normal.	Increased.
(c) Proteins.	Normal.	Reduced.	Normal.	Reduced.

Complete recovery is quite common in the acute stage and is possible in the subacute stage if the disease is of less than one year's duration. In the chronic stage, recovery is not possible but the patient may remain in a state of apparent good health with compensatory polyuria if his diet and activities are restricted within the functional limits of the kidneys and the heart.

Prognosis.—At any stage signs of bad prognosis are:—

(*a*) High blood urea and N.P.N.—especially with a low protein diet. (*b*) Hypertensive neuro-retinopathy. (*c*) Persistent high blood pressure (especially above 200 mm systolic). (*d*) Persistent hæmaturia. (*e*) Signs of heart failure.

Diagnosis.—In the acute stage diagnosis is easy in typical cases with œdema, hypertension, hæmaturia and the characteristic urinary findings. When the onset is insidious or when œdema and hypertension are inconspicuous, diagnosis is difficult unless urine is examined in suspected cases.

Albuminuria and hæmaturia also occur in acute focal nephritis. But there is no œdema nor hypertension in this condition.

The œdema of acute or subacute nephritis is to be distinguished from other causes of generalised œdema (see page 127).

Amyloid nephrosis may also resemble subacute or chronic nephritis. Presence of a prolonged suppurative process in the body and evidence of amyloid disease in liver and spleen or of diarhœa due to intestinal amyloid disease help in the diagnosis.

The diagnosis of chronic nephritis from essential hypertension has already been mentioned. (see page 61).

Congenital polycystic kidneys may give rise to hypertension, albuminuria, hæmaturia and renal failure, and resemble chronic nephritis. The palpation of enlarged kidneys and X'ray of the kidneys confirm the diagnosis.

Treatment.—Treatment of nephritis in all stages consists mainly of: (*i*) Deitetic measures according to the renal function. (*ii*) Symptomatic relief. (*iii*) Prevention of acute recrudescence and avoidance of complications.

Acute nephritis.—Strict rest in bed and protection from cold must be enforced even in mild cases for at least 3 weeks.

Diet should be free from proteins in the initial stages to avoid all strain on the acutely affected kidneys. Carbohydrates in the form of barley water, glucose drinks, fruit juice are given for the first few days. Then (after a fortnight) jelly, biscuits, bread and butter may be added. With improvement in the condition as shown by disappearance of œdema, hypertension and hæmaturia, proteins may be added gradually, beginning with ½ gm. per lb. of body weight. Full protein diet should not be given until albuminuria disappears.

Fluids should be restricted to 1½ pints (a pint in children) a day in the early stage. A daily fluid chart (showing amount of fluid taken and amount of urinary out put) will help in the control of

fluids. If signs of renal failure are present, fluids should be given freely inspite of œdema.

Common salt is avoided until œdema disappears. For œdema adequate measure described before (see page 129) should be taken. Strong diuretics and high protein diet are contraindicated.

If hypertension with its acute symptoms like enecphalopathy or left ventricular failure is present, treatment as in cases of essential hypertension should be adopted. (See page 64).

During convalescence, patient should be kept in bed until albuminuria disappears. Improvement in general health and anæmia should be secured with vitamins and iron.

Obvious septic foci, if present in the nasopharynx, should be eliminated.

Subacute nephritis.—Restriction to bed and protection from cold must be done as before. General hygienic measures of the mouth, bowels and the skin should be taken.

Diet.—This should be rich in proteins and carbohydrates but low in fat. When obvious hypoproteinæmia is present, high protein diet (120 to 220 gms in 24 hours) is indicated.

Contraindications of high protein diet.—(*a*) Red blood cells in centrifuged urine (if more than 4 per field). (*b*) High urea and N.P.N. (*c*) High blood pressure. (*d*) Evidence of impaired renal function, *e.g.*, low specific gravity of urine under conditions of concentration tests. When the above signs are present, diet should be poor in proteins. Prolonged protein starvation, however, may lead to anæmia, tissue wasting, lowered resistance to infection and further hypoproteinæmia. Therefore, minimum proteins to maintain nitrogen equiblirium (½ gm. per pound of body weight) should be given, keeping an eye on the blood urea and N.P.N.

Fluid and salt restriction and other measures for the relief of œdema (already described) should be instituted.

Strong mercurial diuretics are to be avoided. Alkalies, urea and sometimes purine derivatives can be used.

Anæmia should be treated with adequate iron therapy. Septic foci, if present, should be eradicated.

Chronic nephritis.—The treatment at this stage is like that in essential hypertension. The diet and activities should be determined by functional conditions of the kidneys and heart.

The general management as regards rest, exercise etc., should be as in hypertension. Exposure to cold should be avoided.

When there is no obvious nitrogen retention in blood and when the specific gravity of urine under fluid restriction reaches at least 1020, moderate protein diet can be given (½ gm per pound of body weight).

Fluid and salt restriction is unnecessary. In the presence of impaired renal function large amounts of fluids may be beneficial.

Heart failure or uræmia when present require appropriate treatment.

CHAPTER IV

URÆMIA

Definition.—Uræmia is a condition of profound metabolic disturbance due to failure of the normal kidney function.

Originally, the term was used in the belief that the symptoms were due to a retention in the blood of the various urinary excretory products specially urea.

Causes of uraemia.—Any condition which causes sufficient destruction or injury to the renal parenchyma will lead to uræmia. The common causes are:—

(1) *Glomerulonephritis*—usually in chronic, occasionally acute and rarely subacute.

(2) *Essential hypertension*—usually in the malignant type. (Malignant nephrosclerosis).

(3) *Acute necrotising nephrosis* —due to severe poisoning with mercury, arsenic or infections like cholera, Weil's disease, yellow fever etc.

(4) *Chronic amyloid nephrosis.*

(5) *Bilateral hydronephrosis.*

(6) *Polycystic kidneys.*

(7) *Bilateral pyelonephritis.*

(8) *Urinary obstruction.*

Pathogenesis.—The long search for a toxic waste product of nitrogenous metabolism which could explain all the manifestations of uræmia, by its retention in blood, proved to be unsuccessful. None of the known urinary waste products or their metabolic precursors could be shown to be responsible for the manifestations. It is now believed that the complexity of the picture of uræmia is due to a diversity of factors arising out of the metabolic disturbances associated with failure of the kidney functions.

When the kidneys fail to excrete acid sodium phosphate, acidosis with a reduction in the alkali reserve of blood occurs. The acidosis is responsible for the dyspnœa, nausea and vomiting by central action.

Retention of phosphates in blood causes a reduction of serum calcium which in turn produces nervous and muscular irritability, muscular twitchings, tetany and possibly the hæmorrhagic signs.

When the kidneys fail to excrete urea and other nitrogenous waste products, there is an attempt at vicarious excretion of these products through the gestro-intestinal tract, as the saliva, gastric and intestinal secretions. Bacterial decomposition of urea in the mouth is responsible for the ammoniacal odour of breath in uræmic patients. Ammoniacal irritation and excretion of unknown toxic metabolites

are believed to be responsible for enteritis and colonic ulcerations causing diarrhœa or dysentery. A condition of gastritis is also present causing anorexia, nausea and vomiting. The last two symptoms are also partly of central origin due to acidosis and toxæmia.

Retention of products of intestinal putrefaction such as phenols, indican and aromatic oxyacids, which are normally excreted in the urine, may also play some part in the production of uræmic coma by their toxic action on the central nervous system.

Arterial hypertension which very often accompanies most of the kidney diseases causing uræmia produces the acute cerebral manifestations by causing cerebral arterial spasm (hypertensive encephalopathy). These symptoms are therefore not truly uræmic, as they do not depend on renal failure but on hypertension. Cases where these symptoms are prominent were described as acute convulsive uræmia or pseudouræmia in the past.

Lastly it is possible that the dehydration of the tissues partly due to compensatory polyuria, vomiting and diarrhœa, and partly due to increased molecular concentration of the blood and tissue fluids, disturbs the delicate physicochemical processes of vital cellular activity deranging the whole metabolism.

Symptoms and sings.—*Early manifestations.*—The onset is more often insidious than acute. The early manifestations are headache, insomnia, mental and physical lethargy, anorexia, nausea, vomiting, diarrhœa and dysentery, nervous and muscular hyperirritability dimness of vision and pruritus.

Convulsions, temporary paralysis of limbs or face, aphasia and sudden blindness etc., are rarely seen due to associated hypertension.

In the presence of some or all of the above symptoms, urinary and blood changes described below will confirm the diagnosis of uræmia.

Late manifestations.—The patient is emaciated and pale with dry skin and in a drowsy semiconscious or comatose condition with often delirium and hallucinations. Breathing is slow and deep (acidotic dyspnœa or Kussmaul breathing) often of hissing character. Cheyne-Stokes breathing may occur. Breath smells of urine (ammoniacal) and there is bleeding from the gums. Petechiæ may be present in the skin. Tongue is dry, coated and in severe cases heavily furred. Stomatitis is common.

Patient complains of intractable headache, even when drowsy, and is troubled with nausea, vomiting or hiccough. Although muscular twitching and tetany may be present, convulsion is rare. In comatose patients, reflexes are lost and pupils are dilated.

Laboratory findings. Urine—Quantity is diminished and the specific gravity is low or fixed round about 1010. Albumin, various casts, red blood cells etc., are present in varying degrees depending on the nature and stage of the kidney disease.

Blood.—Urea and N.P.N. are increased usually above 100 mgs per cent. There is also increase of uric acid, creatinine and phosphates. Chlorides may be diminished in cases of excessive vomiting. Serum calcium and the carbon dioxide carrying capacity of plasma are lowered.

Renal function tests.—Marked reduction in the renal efficiency is present. The urea clearance test is usually below 10% of the normal.

Complications.—(1) Pericarditis. (2) Cardiac failure with or without varying grades of heart-block. (The so-called uræmic asthma is due to paroxysmal nocturnal dyspnœa of left ventricular failure). (3) Secondary infection like, pneumonia, bronchopneumonia, pleurisy, meningitis, peritonitis, bacillary dysentery.

Diagnosis.—Symptoms such as persistent headache, insomnia, mental apathy, muscular weakness, anorexia, flatulent dyspepsia and diarrhœa in patients with known kidney disease or in elderly subjects should arouse the suspicion of uræmia. The diagnosis can be confirmed by urinary and blood examinations.

Differential diagnosis.—Uræmia has to be differentiated from cerebral vascular lesions, diabetic coma, cholæmia and extra-renal azotæmia. The diagnosis is not always possible on clinical grounds alone and laboratory aid is frequently necessary. The main diagnostic features are shown below.

1. *Cerebral vascular lesions.*—(*a*) *Cerebral haemorrhage and thrombosis*—In both these conditions onset is sudden in apparantly healthy individuals with symptoms of hypertension. A hemiplegia is always present. Nausea, vomiting, diarrhœa, hiccough, furred tongue and bleeding gums etc., are absent. Rise of temperature is unusual in uncomplicated uræmia. C.S.F. contains blood in cerebral hæmorrhage. Urine may contain albumin and various casts due to associated nephrosclerosis or due to hypostasis. Blood urea and N.P.N. are normal or only slightly raised. (*b*) *Hypertensive encephalopathy* (see page 59). (*c*) A widespread involvement of the cerebral vessels by senile atheromatous changes may lead to a diffuse softening of the cerebral substance (*encephalomalacia*) in old people leading to various symptoms resembling uræmia, such as headache, mental and muscular lethargy, mental confusion, etc. The blood pressure is low. Some focal signs of loss of cerebral function due to thrombotic attacks may be present such as, monoplegia, aphasia, facial weakness, hemiparesis, dementia etc. Gastro-intestinal symptoms are absent. Urine may contain albumin due to atheromatous kidney changes. Blood urea and N.P.N. are normal.

2. *Diabetic coma.*—The onset is gradual like uræmia and may be preceded by muscular spasms, abdominal pain, nausea, vomiting, drowsiness gradually deepening into coma. Deep and slow breathing is present but the breath smells of acetone. Mouth shows no bleeding or ulcer and the tongue is clean and red (raw beefy

tongue). Muscles are flaccid, pulse is feeble and rapid. Blood pressure is low and temperature subnormal. Eye balls are soft. Urine contains sugar and acetone. Albumin and some casts may also be present. Blood sugar is high and urea, N.P.N. normal.

4. *Cholaemia* or *acute hepatic failure.*—The onset is acute with headache, delirium, twitchings, convulsions, drowsiness or coma. Deep jaundice is usually present. Hæmorrhages from the various orifices and under the skin are common. Local signs of acute liver damage such as enlargement, tenderness, rapidly diminishing size ascites etc., may be present. Urine contains albumin, blood, leucine and tyrosine crystals. Blood shows high urea, N.P.N. and bilirubin.

5. *Extra-renal azotaemia.*—Conditions in which a failure of the renal excretion occurs due to extra-renal factors has been mentioned already (see page 117). In these conditions oliguria is marked and the specific gravity of whatever urine produced is high. An obvious extra-renal cause is present. Signs of dehydration and hæmoconcentration are present. Blood pressure is low or normal. Blood chloride is low due to excessive loss from vomiting and purging.

Treatment.—The principles of treatment are:—1. Reduction of the work of the kidneys by restriction of proteins in the diet. 2. Correction of altered blood chemistry such as dehydration, acidosis. hypochlorræmia, hypocalcæmia etc. 3. Promotion of excretion through the bowels, skin and kidneys. 4. Symptomatic relief.

1. Complete restriction of proteins in the diet should be done in urgent and acute cases. But in chronic and prolonged cases, low to moderate protein diet should be given to prevent tissue wasting and loss of resistance. Thus 20 to 40 gms. of proteins per day may be allowed depending on the urgency of the symptoms and blood urea and N.P.N. in chronic cases. The rest of the diet consists of carbohydrates and fats.

The diet need not be salt-free except when the blood chloride is high.

Fluid should be given freely, even in the presence of œdema. But as water excretion function of the kidney is also impaired, too much fluid should not be forced specially when there is cardiac failure, as the resulting hydræmia, increases the work of the heart.

In the presence of severe vomiting or in comatose patients 50 cc of a 25% solution of glucose is given intravenously, two or three times a day.

2. Alkalies by mouth or by rectal or intravenous route serve to combat acidosis and improve urinary flow. In acute anuria in cases of cholera or other extra-renal causes 2 to 4% sodi bicarb in normal saline intravenously restores fluid and salt loss, combats acidosis and promotes urinary flow.

In uræmia due to alkalosis acid sodium phosphate instead of alkalies is to be given by mouth.

3. Eliminaton through bowels.—Moderate quantities of nitrogenous waste products can be eliminated through the bowels. Therefore purgatives preferably saline purgatives should be given in sufficient doses to produce one or two fluid motions a day. When diarrhœa is already present, it should not be checked.

Elimination through the skin is only negligible. Alkaline diaphoretics may be given but vigorous diaphoretic measures should be avoided.

Elimination through the kidneys—In cases of chronic kidney disease or in the presence of acute nephritis it is useless to try to stimulate the kidneys by strong diuretics. Alkalies, fluids and glucose intravenously should only be used in such cases to improve urinary secretion.

In cases where cardiac failure is present, urinary secretion improves by adequate measures which improve the cardiac function like digitalis, rest and venesection.

In anuria associated with cholera, blackwater fever etc., intravenous saline infusions with alkalies often restore urinary flow. Counter-irritation in the loins by linseed poultices or dry cupping may also succeed. Injection of caffein sodi benzoas (5 grs.) is also helpful in such cases.

4. Symptomatic relief—(*a*) *Headache, insomnia* and *restlessness*—Sedatives like bromides, chloral hydrate, phenobarbitone, asipirin etc., may be used. Sometimes headache is very resistant and morphine may be required. Lumbar puncture may relieve headache in some cases.

(*b*) *Muscular twitching.*—Intravenous injection of 5 cc of a 10% solution of calcium gluconate relieves muscular twitchings immediately.

(*c*) *Convulsive attacks.*—Where these are due to hypertensive encephalopathy, the treatment should be as in this condition. (See page 64).

(*d*) *Nausea* and *vomiting.*—Being mostly toxic and central in origin, adequate alkalies and fluids may give some relief. Often a condition of gastritis is also present, and local gastric sedatives may be given.

(*e*) *Hiccough.*—This is often very intractable and may require morphine and atropine for relief.

(*f*) *Haemorrhages.*—Large doses of vitamin C (300 to 500 mgms) intramuscularly have been seen to be effective in controlling bleeding in uræmic cases.

CHAPTER V

HÆMATURIA

Presence of blood in the urine is called hæmaturia. The red blood cells are seen intact on microscopical examination of the urinary deposits. This must be distinguished from hæmoglobinuria when no intact red blood cells are seen although chemical and spectroscopic tests for blood pigment in the urine are positive due to presence of hæmoglobin or its derivatives oxyhæmoglobin and methæmoglobin.

Causes of haematuria.—Blood is added to the urine by extravasation somewhere in the kidney or urinary passages either due to a general hæmorrhagic state or due to local lesions. The common causes are the following:—

I. *Causes associated with general haemorrhagic tendency in various parts of the body.*—

(1) Essential thrombocytopenic purpura hæmorrhagica.
(2) Hæmophilia.
(3) Acute leukæmias, (Lymphatic or Myeloid).
(4) Scurvy.
(5) Hæmorrhagic type of acute exanthematous or acute infective fevers, e.g., small pox, measles, Weil's disease, yellow fever.
(6) Essential hypertension.

II. *Causes in the kidney.*—

(1) Acute glomerulonephritis.
(2) Renal calculus.
(3) Tuberculosis of the kidneys.
(4) Malignant growths *e.g.,* hypernephroma (adenocarcinoma), embryoma (Wilm's tumour).
(5) Injury.
(6) Infarction.
(7) Polycystic disease.
(8) Nephroptosis.
(9) Drugs—sulphanilamides, hexamine, cantharides, carbolic acid etc.

III. *Causes in the renal pelvis.*—1. Calculus. 2. Pyelitis (acute). 3. Papilloma. 4. Angioma.

IV. *Causes in the ureters.*—1. Impaction of calculus.

V. *Causes in the urinary bladder.*—1. Calculus. 2. Acute cystitis. 3. New growths, e.g., papilloma, carcinoma. 4. Tuberculosis of the bladder. 5. Bilharziasis (schistosomiasis). 6. Injury. 7. Filariasis.

VI. *Causes in the prostate.*—1. Senile enlargement. 2. Adenoma 3. Carcinoma. 4. Tuberculosis. 5. Congestion. 6. Abscess.

VII *Causes in the urethra.*—1. Acute urethritis. 2. Injury. 3. Impacted calculus. 4. Caruncle.

Investigation and diagnosis. 1. *Age.*—Common causes of hæmaturia at different ages are

(*a*) In new born baby—Hæmorrhagic diseases of new born, due to vitamin K deficiency—(more commonly causes melæna).

(*b*) In childhood—(*i*) General hæmorrhagic states like lymphatic leukæmia, acute infective fevers, hæmophilia or scurvy. (*ii*) Local causes may be stone in the bladder, acute nephritis, pyelitis etc., and rarely Wilm's tumour (embryoma) of the kidney.

(*c*) In young adults—(*i*) Renal calculus. (*ii*) Tuberculosis of the kidney. (*iii*) Gonococcal urethritis.

(*d*) In elderly patients—Calculus, hypernephroma, other malignant tumours, enlarged prostate, hypertension etc.

2. *Sex.*—In females besides other causes a caruncle at the external urethral orifice may often cause hæmaturia.

3. *Nature of haematuria.*—(*a*) *Quantity of blood*—Profuse hæmaturia is commonly due to (*i*) tumours in the kidneys or bladder, (*ii*) injuries with rupture of the kidney, (*iii*) rarely in tuberculosis of kidney, (*iv*) rarely in prostatic enlargement.

(*b*) *Relation to the act of micturition*—(*i*) Bright blood at the beginning of micturition indicates bleeding from prostate or urethra. (*ii*) Blood at the end of micturition indicates bleeding from bladder. (*iii*) When blood is intimately mixed with urine (smoky urine) bleeding is from the kidneys or renal pelvis.

(*c*) *Exciting cause*, if any—such as injury to the back or abdomen causing rupture of the kidney. When hæmaturia follows excessive movements or joltings etc., it is probably due to renal calculus. Instrumentation of urethra may also cause hæmaturia from local injury.

4. *Other associated symptoms with haematuria.* (*a*) Pain—(*i*) Colic - unilateral spasmodic pain starting from the renal angle (angle between the 12th rib and outer border of the erector spinae muscle) and passing forwards to the groin may be due to passage of blood clots or stones from the renal pelvis to the bladder. The seat of bleeding can be localised in the kidney in such cases and if history of similar colics in the absence of hæmaturia are present, renal calculus can be diagnosed. (*ii*) Lumbar pain—dull aching pain in the renal angle suggests a renal cause of hæmaturia. Such pain may occur in various kidney lesions. (*iii*) Pain at the tip of the penis—specially after micturition indicates irritation at the trigone of the bladder. (*iv*) Sacral pain—suggests malignant disease of bladder or prostate. (*v*) Hypogastric pain indicates cystitis. (*vi*) Painless hæmaturia—this is common in tuberculosis and benign tumours in the urinary tract.

(*b*) Increased frequency of micturition—This may occur due to local causes in the bladder or in acute pyelitis and tuberculosis of the kidney.

(*c*) Hæmorrhage elsewhere in the body, such as hæmoptysis (coughing out blood), hæmatemesis (vomiting of blood), melæna (altered blood in fæces), epistaxis (bleeding from nose), bleeding from gums, purpura (hæmorrhagic spots in the skin), etc.

This will suggest a general hæmorrhagic state due to one of the general causes.

(*d*) Constitutional symptoms with fever, malaise, rigors etc., indicate an infection either local, as in pyelitis, cystitis, tuberculosis. General cutaneous rashes or eruptions will be present in hæmorrhagic small pox or measles.

5. *Examination of the urogenital system*—(*a*) Palpation of the kidneys—Enlargement in size, if unilateral, may be due to tuberculosis, hypernephroma, hydro or pyo-nephrosis, pyelonephritis etc.

Bilateral enlargement is common in polycystic disease, Wilm's tumour and bilateral hydronephrosis.

Tenderness over the kidneys or at the renal angle may occur in renal stone or other kidney diseases and pyelitis.

(b) Palpation over the bladder—Tenderness may be due to acute cystitis. Distension if present may be due to prostatic enlargement.

(c) Inspection of the external genitals and urinary meatus for local causes.

(*d*) Testis and epididymis—Evidence of tuberculosis in these organs may be due to a tuberculous disease of the kidneys or bladder.

(*e*) Rectal and vaginal examination—Rectal examination in males will reveal any enlargement or tenderness of the prostate. If there is marked irregularity, hardness and fixity of the prostate, malignant tumour is diagnosed.

The base of the bladder may also be felt by rectal or vaginal examination. Thickening and induration of the bladder base with fixation on the surrounding structures, indicate vesical carcinoma.

The openings of the ureters in the bladder should also be palpated. Thickening and induration at the lower ends of the ureters, are early signs of tuberculosis of the bladder.

6. *A general examination of the patient and examination of other systems should be done for evidence of the various general haemorrhagic diseases.*—Evidence of bacterial endocarditis or other sources of emboli should be looked for in cases of suspected infarction.

7. *Examination of urine.*—Presence of large number of pus cells along with red blood cells indicates pyelitis, pyelonephritis, tuberculosis of kidneys or cystitis. Secondary infections causing pyuria may also complicate renal calculus or growths.

Excess of crystals of uric acid, urates, oxalates etc., with blood, may indicate presence of stones.

Occasionally portions of growths may be passed in the urine in villous papilloma or carcinoma of the bladder.

Some information as to the site of bleeding may be obtained from the shape of blood clots present in the urine.

Presence of excess of albumin with hyaline, epithelial and blood casts indicates acute nephritis.

The deposits should be examined for M. tuberculosis and cultured for Bact. coli and other organisms which cause pyelitis and cystitis.

Sometimes gravels (calculi) are passed in urine in cases of urinary calculi. Enquiries should be made for such a history.

8. *Examination of blood and other investigations are to be made* to confirm diagnosis of suspected general hæmorrhagic diseases, such as—blood count in leukæmia, platelet count, coagulation and bleeding time in purpura and hæmophilia.

9. *X'ray examination of the kidneys and bladder.*—Presence of calculi is revealed by X'ray examination. Gross enlargement or deformity of the kidneys may also be visible indicating growths, tuberculosis, hydronephrosis etc.

Intravenous pyelography is more helpful in doubtful cases. Deformity of the calices of renal pelvis indicates presence of growth.

In tuberculous kidney, there may be evidence of deficient excretion through the affected kidney, and irregularity or indefinite margins of the calices may be seen.

10. *Cystoscopy.*—Evidence of local disease in the bladder can be obtained by a cystoscope. When no local changes are seen in the bladder, evidence of bleeding from kidneys may be seen by escape of hæmorrhagic urine from one or both of the ureteric orifices.

Ureteric catheter may be passed and urine collected from each ureter to determine which kidney is affected. Retrograde pyelography may also be done by introducing dyes into the ureters to fill up the renal pelvis, and taking x'ray pictures subsequently.

HÆMOGLOBINURIA

When hæmoglobin and its derivatives, instead of the red blood cells, escape in the urine, the condition is called *haemoglobinuria.*

The urine may be slightly reddish or blood-red, dark brown or black in colour depending on the severity of hæmoglobinuria. It also contains excess of proteins, granular pigments, casts etc.

Hæmaturia is distinguished by absence of red blood cells in the deposits and bilirubinuria by negative tests for bile pigments. The hæmoglobin and its derivatives, oxyhæmoglobin and methæmoglobin are identified by spectroscopy.

Causes.—Hæmoglobinuria is always due to excessive hæmolysis in the general circulation by hæmolytic agents. The free hæmoglobin

is then excreted by the kidneys as a foreign protein. The causes are therefore always extrarenal. Common causes are—1. Blackwater fever. 2. As a sequel to blood transfusion, (*a*) due to heterogenous blood groups in the donor and recipient; (*b*) in repeated transfusion from same donor of homologous blood group due to isoagglutinins. 3. Infection with Cl. welchii, subtertian malaria, syphilis. 4. Paroxysmal hæmoglobinuria. 5. Snake poisoning. 6. Certain drugs and poisons—potassium chlorate, arseniuretted hydrogen, nitrites, phenylhydrazine, turpentine, quinine, saponin, poisonous mushrooms etc. 7. In some functional neurovascular disturbances *e.g.*, Raynaud's disease, angioneurotic œdema.

PYELITIS AND PYELONEPHRITIS

Infection of the renal pelvis by micro-organisms is called pyelitis. When it is associated, as is frequently the case, with foci of infection in the kidney substance the term pyelonephritis is used.

Aetiology.—*Age*—It may occur at any age. *Sex*—Amongst adults, females more commonly affected than males; in infancy girls are more affected; in childhood and in old age—over 50 years—males and females are equally affected.

Infecting organisms.—Most commonly Bact. coli. Less commonly Staphylococcus aureus, Streptococcus fæcalis, Bact. proteus, Ps. pyocyanæ, Bact. typhosum, etc.

Mode of infection.—(a) *Haematogenous*- Bacilli enter the circulation from the intestines or other foci of infection, and in the process of elimination through the kidneys, cause infection, specially when some local predisposing cause is present. Bacilluria may however occur for short periods without local inflammation. (b) *Ascending infection.*—Infection from lower urinary organs such as urethra, bladder, prostate etc., may ascend in the lumen of the ureters when there is urinary obstruction. (c) *Lymphatic route.*—Infection from lower urinary organs or from the intestines may be carried by lymphatic channels.

Predisposing causes.—(a) Urinary obstruction—by such conditions as urethral stricture, enlarged prostate, impacted stones in the ureters, pressure on ureters by external growths or aberrant renal arteries, stones in the renal pelvis etc. (b) Local kidney diseases—as tuberculosis, new growths, calculi, hydronephrosis etc. (c) Pregnancy. (d) Intestinal conditions favouring bacilluria, eg. enteritis, intestinal ulcers, enteric fever, colitis, appendicitis, intestinal stasis etc.

Pathology.—There is catarrhal inflammation in the renal pelvis. Foci of infection in the kidney substance to a greater or less extent are commonly present and small abscesses may be found. In severe cases kidney is swollen, and wedge shaped abscesses are present, specially in cases of ascending infection in urinary obstruction.

Signs and Symptoms.—See Table XI.

Diagnosis.—In cases with local symptoms and signs, diagnosis is easily made by examining the urine for pus cells and culturing for micro-organisms. Urine must be collected by catheter or with reasonable precautions against external contamination. Presence of bacilli alone without pus cells should be regarded as harmless bacilluria. In cases of obscure pyrexia urine should be examined and cultured to exclude chronic pyelitis. It must be remembered that Bact. coli or other infections of the urinary tracts may be secondary to such diseases as tuberculosis of kidneys, renal calculi etc. Investigations to exclude such diseases should be made.

Treatment.—A. In the early stage of high fever and toxæmia. Rest in bed. Large quantities of fluids to keep up a copious urinary

flow. Sufficient doses of alkalies to keep the urine alkaline throughout 24 hours should be given as alkaline urine inhibits the growth of Bact. coli.

Urinary antiseptics should not be given at this stage as they require, (a) fluid restriction to obtain sufficient concentration of the drugs in urine. (b) administration of acid salts to render the urine sufficiently acid (except in cases of sulphanilamides) for their optimum action. Both these conditions are badly tolerated in the presence of high fever.

Diet should be liquid; preferably without milk and meat products, to reduce intestinal bacterial putrefaction.

Bowels should be kept open by calomel and saline purgatives.

B. When temperature has subsided or has been reduced by alkalies for the first 3 or 4 days—urinary antiseptics should be used to sterilise the urine.

Urinary antiseptics.—(i) Sulphanilamide and its derivatives like sulphapyridine, sulphathiazole or sulphadiazine. These are effective against B. coli and Bact. proteus infections but are of little value in Ps. pyocyanæ or Streptococcus fæcalis infections. Sulphathiazole alone is effective against Staphylococcus aureus. These drugs act better in alkaline urine, but require sufficient concentration in urine to be effective. The dose is 2 gms. to start with, and 1 gm. every 4 hours for 2 to 3 days; and then 1 gm. every 6 hours for another 3 to 5 days. The patient should be watched for toxic signs like cyanosis due to sulphæmoglobinæmia or methæmoglobinæmia, leucopenia, hæmaturia etc.

(ii) Mandelic acid and its derivatives—Mandelic acid, sodium, ammonium or calcium mandelate are bactericidal to Bact. coli when the urine is strongly acid (5 to 5.5 pH). It is necessary therefore to give acid sodium phosphate or ammonium chloride in sufficient doses to keep the urine at the required pH, as shown by a pink colour with methyl red indicator. Various proprietory preparations, such as Neoket or Mandecal contain mandelic acid salts and acidifying agents. 12 gms of mandelic acid or its salts should be given daily (3 gms, 4 times a day) for 7 to 10 days. Fluid restriction is necessary, and urine passed every time should be tested for correct acidity. Mandelic acid should not be given in the presence of impaired kidney function or hæmaturia.

(iii) *Hexamine.*—Hexamine acts as urinary antiseptic by liberating formaldehyde in acid urine. The urine should be rendered acid by acid sodium phosphate 15 to 20 gr. 3 times a day, after meals. Hexamine 10 to 20 gr. is given before meals (3 times a day).

In persistent cases acid and hexamine treatment may be alternated with periods of alkalies.

C. Vaccines—Autogenous vaccines are sometimes useful in chronic cases.

APPENDIX A

ELECTROCARDIOGRAPHY

The origin and spread of the cardiac impulse of excitation and contraction in the auricles and ventricles excite an electric current. This can be detected by a sensitive galvanometer connected with electrodes placed at different parts on the surface of the body. A modification of the Einthoven's string galvanometer is used for this purpose. The deflections of the string can be recorded on a moving photographic plate by arranging a light in such a way as to throw a shadow of the string on the plate. A permanent record of the electrical variations associated with each heart-beat can thus be obtained and such a record is called an *electrocardiogram*.

Leads—Although electrodes connecting any two parts of the body with the galvanometer may detect the electric currents of cardiac contraction, certain connections have been standardised for universal use in routine electrocardiography. These connections are called 'Leads'. Originally three leads were in routine use. They are now called the 'classical leads'. These are:—

Lead I—Connecting the right and the left forearms. (Transverse).

Lead II—Connecting right forearm and left leg. (Axial).

Lead III—Connecting left forearm and left leg. (Left lateral).

Recently two more leads have come into use. These are called 'chest leads' because one of the electrodes is placed on the chest over the præcordium. These are:—

Lead IV R—Connecting a præcordial electrode (placed at the outer border of the apex-beat) and the right forearm.

Lead IV L—Connecting a præcordial electrode and the left forearm.

The Electrocardiogram.—The galvanometer string at rest records a dark broad line forming the base line, and its deflections are recorded as waves above or below the base line. The amplitude of the waves, that is, their height or depth from the isoelectric base line, is measured in millimeters by horizontal lines in the electrocardiogram, each millimeter representing 0.1 millivolt. The width of the waves represents the duration of each defelction and is measured by vertical lines in the electrocardiogram, the darker lines representing periods of 1/5 seconds. (See fig. 37.)

Deflections or waves in a normal electrocardiogram.—There are five deflections during each cardiac systole. These deflections or waves are named as P, Q, R, S, and T waves.

The P wave occurs during auricular systole. Q, R, S and T waves occur during ventricular systole and are together known as

ventricular complex. P, R and usually the T waves are upward or positive deflections, while the Q and S waves are downward or negative deflections.

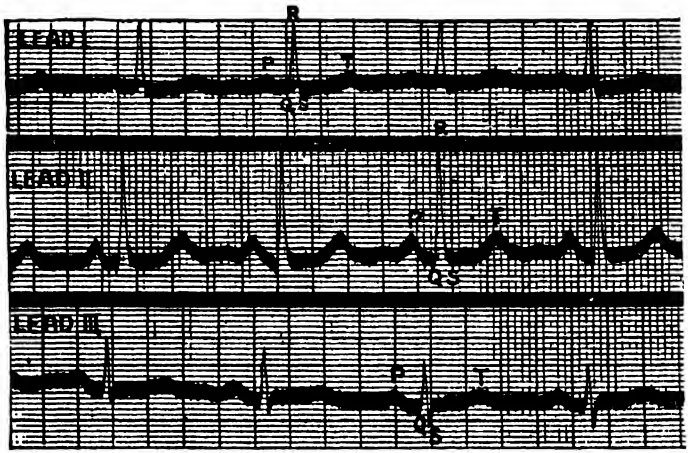


Fig. 37. Normal electrocardiogram.

Significance and character of the waves in different leads under normal and abnormal conditions.—P wave—This is due to auricular systole. It is upright (in all the leads), blunt, varies in amplitude from 1 to 3 mm and has a maximum duration of 0.1 sec.

A large P wave indicates auricular hypertrophy. Inverted P waves (See Fig. 19, page 76) indicate abnormal origin of the cardiac impulse (i.e., outside the sino-auricular node). In auricular fibrillation P waves are replaced by irregular small and very rapid oscillations with very irregular QRS complexes (See page 84, Fig. 26). In auricular flutter or auricular paroxysmal tachycardia rapid but regular small oscillations occur 2, 3 or 4 times more frequently than the QRS complexes (See Fig. 27, page 85).

QRS Waves.—These three waves occur in rapid succession and are described together as QRS complex. The Q wave is a slight downward deflection which is often absent normally. It is followed by a sharp spiky upward deflection—the R wave, and then a smaller sharp downward deflection—the S wave. The later is however not constantly present. Of all the waves R has the maximum amplitude. The duration of the QRS complex does not exceed 0.1 second. The QRS complex represents the spread of the cardiac impulse through the arborisations of the bundle of His and is called the initial ventricular deflection. In the chest leads the QRS complex is biphasic with equal upward and downward deflections. The QRS deflections are followed by an isoelectric period called the S-T interval. In

leads I and III the QRS waves are normally of smaller amplitude than in lead II.

The QRS waves vary greatly in amplitude under normal conditions. But marked low voltage of these waves occurs in gross myocardial diseases, ventricular dilatation or in hypothyroidism and constrictive pericarditis.

Notching or slurring of the R or S waves—when marked indicate myocardial disease with intraventricular block (See Figs. 24 and 25, pages 81 and 82).

Abnormally shaped QRS complexes:—When QRS complexes are abnormally wide, deep, monophasic, 'M' or 'W' shaped, they may be due to the following conditions:—(a) Extrasystole—The abnormal complex occurs as an isolated abnormality in the course of normal complexes (See Fig. 18, page 75). (b) Intraventricular block—Abnormal complexes occur regularly with each heart-beat and in normal relation with the preceding P waves (See Figs. 24 and 25). (c) Ventricular paroxysmal tachycardia—Abnormal complexes occur in very rapid succession independent of the P waves.

P-R interval.—The interval between the beginning of P wave and the beginning of the QRS complex represents the time for conduction of the cardiac impulse from the auricles to the ventricles by the auriculo-ventricular bundle. This should not exceed 0.2 sec. normally.

In early stages of heart-block the P-R interval is prolonged (See Fig. 21, page 78). In auriculo-ventricular nodal rhythm—the P-R interval is shortened. The P waves in these cases, being the result of impulses from the A-V node in a reverse direction, are always inverted and may occur immediately before or after or simultaneously with the QRS waves. In complete heart-block there being no constant relation between the P waves and QRS complexes, P-R intervals are extremely variable. (See Fig. 23).

T Wave.—This is the final ventricular deflection and is due to the retreat or subsidence of the cardiac impulse in the ventricular muscle. It is a blunt wave about 3 to 4 mm high and has a duration of 0.2 to 0.3 seconds. It is usually upright in all leads but may be normally flat or inverted in lead III. In chest leads it is high and upright.

Inversion of the T waves occurs usually in coronary insufficiency or in coronary thrombosis. In apical infarction the T wave in lead I (T_1) is inverted and upright in lead III. In basal infarction T_3 is inverted, T_1 being upright. Inversion of T waves may also occur in all the leads in digitalisation, in myocarditis due to infections or in myxœdema. In chest leads T wave is inverted in coronary thrombosis or insufficiency (See Fig. 15, page 71). In intraventricular block or in ventricular extrasystole the T wave is usually in opposite direction to the main deflection of the QRS complex.

S-T Segment.—This is an isoelectric period between the end of S (or R when S is absent) and the beginning of T waves. Elevation or depression of this segment occur in early stages of coronary thrombosis. Later (after a few weeks) it tends to become normal although the T wave (which is in a direction opposite to the S-T deflection) changes persist longer. The deviation of the S-T segment occurs in opposite directions in leads I and III. In apical infarction (T_1 type) S-T is elevated in lead I and depressed in lead III. In basal infarction (T_3 type) the changes are reversed in leads I and III (See Fig. 14, page 69).

S-T segment is also depressed in all the leads in digitalisation.

Axis deviation and Left or Right ventricular preponderance.—When the maximum deflections of the QRS complexes point away from each other in the 1st and 3rd leads (that is, a prominent R_1 with a prominent S_3), a left axis deviation is present. On the other hand if the maximum deflection of QRS complex is downward in lead I and upward in lead III (a prominent S_1 and a prominent R_3) a right axis deviation is present.

A left axis deviation, when marked, indicates preponderating hypertrophy of the left ventricle (left ventricular preponderance); similarly a right axis deviation indicates right ventricular hypertrophy (right ventricular preponderance). It should be noted, however, that a transverse position of the heart such as due to a raised diaphragm may produce a left axis deviation. Similarly a more vertical heart with a lowered diaphragm will cause right axis deviation.

APPENDIX B

EXAMINATION OF URINE

Collection of specimen.—For all routine examinations, the first urine in the morning is most suitable because little or no fluid is ingested for a number of hours at night, thus approaching to certain extent the conditions for concentration test. It is also free from any orthostatic albuminuria. For special purposes other samples of urine may be necessary, as in cases of early diabetes, a sample of urine passed 2 or 3 hours after a meal may show sugar, when the fasting sample is sugar-free. For quantitative estimations, 24 hours' collection is necessary. The urine should be collected in a clean bottle. In females the genitals should be cleaned before micturition as contamination of urine with vaginal discharge may cause false evidence of albuminuria and pyuria. The examination should be done soon after the urine is voided. On keeping the urine undergoes ammoniacal decomposition by bacteria; the urea being converted into ammonia. The reaction changes to alkaline side, and phosphates are precipitated. Organised deposits like pus cells, casts etc., also undergo decomposition on prolonged keeping.

For all bacteriological examination the urine should be collected by catheterisation of the bladder. Midstream urine in case of males, if collected after properly cleaning the glans penis is also reasonably free from external contamination.

Physical examination.—The following are to be noted—1. *Quantity*—measured by a measuring cylinder. (See page 110). 2. *Colour.* (See page 111). 3. *Transparency and deposits if any.* (See page 112). 4. *Reaction*—Blue and red litmus papers are used. On soaking a piece of blue litmus paper in urine it turns red if the urine is acid. Similarly red litmus paper is turned blue if urine is alkaline. 5. *Specific gravity*—This is measured by an instrument called urinometer, which is graduated from 0 to 60 (see fig. 38).

The urinometer is allowed to float in a column of urine in a long cylindrical vessel of sufficient diameter to prevent the urinometer touching its sides. The mark on the graduated scale of the urinometer which is just on level with the surface of the urine is noted. Specific gravity is expressed by adding 1000 to the reading (1000 being taken as specific gravity of water).

Chemical examination.—In the routine examination the following abnormal constituents of urine are tested for:—

(i) Albumin, (ii) Sugar, (iii) Ketone bodies: Acetone and diacetic acid, (iv) Bile pigments and bile salts, (v) Indican, (vi) Urobilinogen and urobilin, (vii) Blood and hæmoglobin.

Albumin.—The common tests applied for the detection of albumin, give positive reaction with other proteins besides albumin which may be present in the urine such as globulin, hæmoglobin and mucin. The term 'albuminuria' therefore really indicates 'proteinuria'.

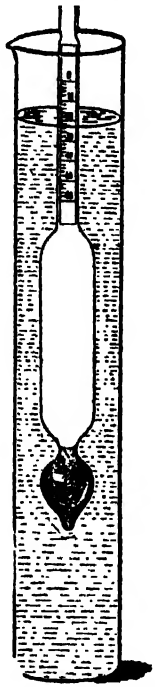


Fig. 38. Urinometer.

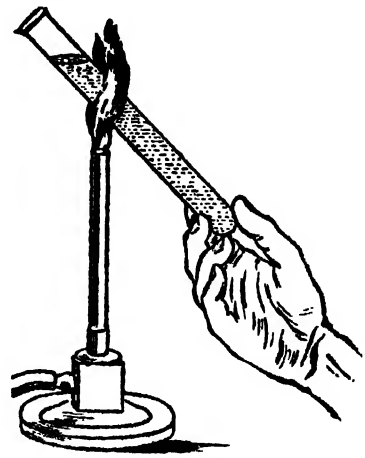


Fig. 39. Boiling test for the presence of albumin in urine.

Although it is possible to distinguish between albumin and globulin, it is of little practical significance. Hæmoglobin and mucin however can be easily distinguished (*vide infra*).

Tests.—1. *Boiling test*—A test tube is filled with clear (if necessary after filtration) urine up to about three-fourth of its length and the upper portion is boiled by holding it in a flame as shown in the figure (Fig. 39). Appearance of a turbidity which persists after addition of a drop or two of acetic acid (33 per cent) is due to presence of proteins.

Precautions.—(a) Urine must be clear, otherwise a slight cloud of proteins may be missed. (b) Acetic acid must always be added after boiling, because the removal of carbon dioxide by boiling renders the urine more alkaline and precipitates phosphates. Acetic acid redissolves the phosphates but precipitated proteins persist. (c) In alkaline urine the proteins may not be precipitated until the acid is added because the metaproteins formed from the proteins on boiling are soluble in alkaline solution, and only precipitated metaproteins can

be coagulated by heat. (d) In strongly alkaline urine 1 or 2 drops of acetic acid may not suffice. In such cases therefore it is better to acidify the urine with acetic acid until it is slightly acid to blue litmus, before boiling. Too much acidification is to be avoided because the metaprotein is soluble in acid and no coagulation occurs on heating. (e) Mucin may be precipitated by acetic acid and remain undissolved in excess of acid. The precipitate however may appear even without heating. (f) During heating a cloud may appear much earlier and disappear at boiling point. This is due to presence of Bence-Jones proteins.

2. *Salicylsulphonic acid test.*—In a column of urine, about 1" high in a test tube, a few drops of saturated solution of salicylsulphonic acid are added. A white turbidity indicates presence of proteins (including nucleoproteins and proteoses). A false positive reaction due to precipitation of uric acid may occur.

3. *Nitric acid ring test.*—Concentrated nitric acid is taken in a test tube to form a column about ½" high and urine is poured slowly by the side of the test tube. A white ring at the junction of two layers is a positive reaction.

Note.—(a) False reactions are given by precipitation of urea nitrate, uric acid, bile acids etc. (b) Normal urinary pigments may be oxidised to dark colour by nitric acid obscuring the white ring. (c) Proteoses, Bence-Jones proteins and mucin also give a positive reaction with this test.

Sugar.—Glucose is the most important sugar whose presence in the urine is sought for.

Tests—1. *Fehling's test*—

Reagents:—

Fehling's solution No. 1—

Crystalline copper-sulphate 34.64 gm.
Conc. sulphuric acid 0.5 cc.
Dist. water to 500 c.c

Fehling's solution No. II—Rochelle salt (Sodium potassium tartarate) 176 gm. is dissolved in 300 to 400 cc of distilled water with heat. Then 77 gm of potassium hydroxide sticks are added and allowed to dissolve. The solution is then cooled and made up to 500 cc.

Test.—Equal quantities of Fehling's solutions No. I and II are mixed to make a column of about 1" in a test tube. The same amount of urine is taken in another test tube. Both test tubes are heated in a flame simultaneously and when boiling they are mixed by pouring the contents of one into the other. Without further heating the mixture is allowed to stand. Appearance of a red or yellow precipitate of cuprous oxide indicates presence of sugar (which acts as a reducing agent).

Note.—(a) Prolonged boiling of Fehling's solution and urine may cause a greenish precipitate due to the action of strong alkalies on

normal urinary constituents. (b) Due to the strong alkalinity of Fehling's solution, small quantities of sugar may be caramelised on heating and rendered ineffective as a reducing agent. Benedict's solution being less alkaline does not cause this and therefore Benedict's test is more delicate. (c) Proteins when present in excess in the urine, are precipitated on heating causing confusion. In such cases the urine should first be boiled to precipitate the proteins and then filtered. The filtrate can be used for testing sugar. Small quantities of proteins cause no difficulty. Phosphates may be precipitated due to strong alkalinity, producing a greyish white deposit at the bottom. (d) Fehling's solution may be reduced by other substances which may appear in urine as: (i) other sugars *e.g.* lactose, pentose, (ii) homogentisic acid which is present in alkaptonuria, (iii) creatinine and uric acid when present in increased concentration in normal urine, (iv) salicyluric acid after administration of salicylates or aspirin by mouth.

2. *Benedict's test*—

Reagent (qualitative)	
Sodium citrate	173 gm.
Sodium carbonate (anhydrous)	100 gm.
Copper sulphate (crystals)	17.3 gm.

The citrate and carbonate are dissolved in 600 cc distilled water with heat, and the copper sulphate is dissolved in 100 cc distilled water. The latter is mixed with the former with constant stirring. When cold the volume is made up to 1000 cc with distilled water.

Test.—5 cc of Benedict's solution is taken in a test tube and 8 drops of urine added. The mixture is then heated to boiling in the flame for 2 minutes. A yellow or red precipitate of cuprous oxide indicates presence of sugar. When sugar is present in very small quantities a green precipitate may form But this may be due to other causes than sugar. A white precipitate indicates phosphates.

A rough estimate of the amount of sugar present in the urine may be obtained from the colour of the precipitate and the supernatant fluid. Thus: (i) Slight yellow precipitate at the bottom, with blue fluid above, on standing....sugar about 0.2%.

(ii) Definite orange precipitate at the bottom with the blue fluid above....sugar about 0·5%.

(iii) Heavy orange-brown precipitate with slight blue colour of fluid above....sugar about 1%.

(iv) Bright red precipitate with clear fluid above....sugar about 2% or more.

Note.—Benedict's solution is also reduced by: (i) other sugars e.g., lactose and pentose, (ii) homogentisic acid. (Due to small quantity of urine used creatinine and uric acid cannot be of sufficient amounts to cause any reduction).

Homogentisic acid can be easily identified by the darkening of urine on standing or alkalinisation.

Lactose is commonly present during lactation or pregnancy and pentose is present in some people as an inborn error of metabolism.

Glucose can be readily distinguished from other sugars in urine by fermentation test with yeast or by other more elaborate chemical tests. For practical purposes however this is unnecessary, as the presence of a reducing substance in the presence of symptoms suggesting diabetes mellitus can be taken as diagnostic. Blood sugar estimation confirms this.

Ketone bodies.—*Acetone and aceto-acetic acid*—Aceto-acetic acid (Diacetic acid) is passed in urine in states of ketosis. In the bladder or on standing after the urine is passed, this is converted into acetone.

Tests.—1. *Rothera's test*—About half a test tube of urine is saturated with ammonium sulphate or chloride and a pinch of powdered sodium nitroprusside is added. The mixture is well shaken. Then 2 or 3 cc of liq. ammon. fort is poured by the side of the test tube. A positive reaction is shown by a permanganate coloured ring at the junction of the two liquids. Saturation with ammonium sulphate removes certain interfering substances and makes the test more sensitive. The depth of the colour and its rapidity of development are measures of concentration of the ketone bodies.

2. *Gerhardt's test.*—To about 1" column of urine in a test tube a solution of ferric chloride (10 per cent) is added drop by drop. At first a precipitate of ferric phosphate develops which dissolves in excess of the reagent. A positive reaction is shown by development of a port wine or mahogany red colour. If excess of ferric chloride is added a positive reaction may be masked by the colour of the reagent.

Note.—(a) Rothera's test is positive in the presence of both acetone and aceto-acetic acid, and is more sensitive than Gerhardt's test. (b) Gerhardt's test gives positive reaction with aceto-acetic acid only, and not by acetone. (c) Gerhardt's test may be positive even in the absence of aceto-acetic acid if the patient is taking drugs like salicylates, aspirin, antipyrin etc. To distinguish such false positive reactions, the test is repeated after boiling the urine for some time. In a true positive reaction the test now becomes negative because the aceto-acetic acid is changed into acetone on boiling. If the reaction is due to drugs, it persists even after boiling. (d) Rothera's test is free from above fallacies as the drugs give a negative reaction.

Bile.—*Test for bile salts—Hay's sulphur test*—Finely powdered flower of sulphur when sprinkled on the surface of urine sinks to the bottom, if bile salts are present.

Tests for bilirubin.—1. *Gmelin's test.*—The urine is filtered several times through a piece of blotting paper, and a drop of strong nitric acid is allowed to fall on the paper in the centre of the soaked area. A play of colours (with blue or green) is seen on the paper, if bilirubin is present.

2. *Iodine test.*—A solution of liq. iodine, diluted with equal quantity of distilled water, when poured by the side of a test tube one-fourth filled with urine, gives a green ring at the junction, if bilirubin is present.

3. *Fouchet's test*—Reagents—(i) 10% Barium chloride solution
(ii) Fouchet's reagent—Trichloracetic acid 259 gm.
Distilled water 100 cc.
Ferric chloride, 10 per cent. 10 cc.

A 2″ column of acid urine (acidified with acetic acid if necessary) is mixed thoroughly with half its volume of barium chloride solution. The mixture is filtered. (If no or little precipitate occurs with barium chloride 1 or 2 drops of saturated ammon. sulph is added before filtering). The filter paper is now spread out and a drop of Fouchet's reagent is allowed to drop on it. A green or blue colour shows the presence of bilirubin.

Urobilin and urobilinogen.—Urobilinogen is normally present in urine in small quantities and it changes to urobilin on standing. Urobilinogen and urobilin are present in excess in cases of increased hæmolysis and when liver cells are damaged.

Tests.—(1) *Ehrlich's aldehyde reaction*—A few drops of a 3 per cent solution of paradimethylaminobenzaldehyde in 50% hydrochloric acid is added to a column of urine about 1″ high in a test tube. A positive reaction is shown by a deep red colour indicating excess of urobilinogen.

(2) *Schlesinger's test for urobilin.*—A few drops of liq. iodine is added to a 1″ column of urine in a test tube. In another test tube about 0.5 gm. of zinc acetate and about 5 cc. of absolute alcohol are taken. The contents of the two test tubes are thoroughly mixed by pouring repeatedly from one into the other. The mixture is then filtered. A compound of zinc with urobilin is formed and gives the filtrate a green fluorescence.

(3) *Spectroscopic test.*—Urobilin gives a characteristic band in the green between C and F lines of the spectrum.

Indican.—Indican (Potassium indoxyl sulphate) is produced in the intestines as a result of bacterial putrefaction and is absorbed to be excreted in the urine. Excess of indican in urine indicates intestinal stasis and putrefaction.

Test.—To a 1″ column of urine in a test tube an equal amount of concentrated hydrochloric acid with 0.2 per cent ferric chloride is added. The mixture is thoroughly mixed by shaking. A little (2 or 3 cc) of chloroform is now added and the mixture shaken again. The chloroform layer at the bottom is coloured blue if indican is present.

If the patient is taking iodides or bromides, the chloroform layer will be coloured reddish-violet or yellowish brown due to liberated iodine or bromine.

Blood.—Blood is best detected by examining the centrifuged

deposits for red blood cells; and free hæmoglobin in urine can be detected by spectroscopy.

Chemical test—Guaiacum test—A column of urine, about 1″ in a test tube, is boiled and after cooling 2 drops of freshly prepared tincture of guaiacum is added. After shaking, ozonic ether (hydrogen peroxide in ether) is poured by the side of the test tube. If blood is present a blue ring forms at the junction of the fluids.

Note.—False positive reaction may be given by pus in the urine or when the patient is taking iodides.

Quantitative estimation.

Sugar.—(1) *With Fehling's solution*—

A burette is filled with urine diluted 10 times with distilled water—(urine 10 cc + distilled water 90 cc). 5 cc each of Fehling's solution No. I and No. II (the same solutions as are used in qualitative tests) are taken in a porcelain basin, and diluted with distilled water. The basin is then heated on a flame. To the boiling Fehling's solution in the basin, diluted urine from the burette is added gradually. Cuprous oxide will be precipitated in the basin and when the solution has been completely reduced, the blue colour will disappear. This is the end point of the titration and no further urine is added. The amount of diluted urine used is read from the burette. The amount of sugar is calculated thus:—10 cc of Fehling's solution requires 50 mgm. (0·05 gm.) of glucose for complete reduction. The amount of fluid run from the burette divided by 10 gives the amount of undiluted urine containing 50 mgm. of glucose. From this the percentage can be easily calculated.

$$\frac{0{\cdot}05 \times 10 \times 100}{\text{Burette reading in cc}} = \text{gms of glucose in 100 cc of urine.}$$

Note.—It is difficult to find out the end point *i.e.*, just when all the blue colour of the Fehling's solution is discharged because of the red precipitate. The basin should be tilted so that the supernatant fluid may be seen against the white background of the basin, away from the red deposit at the bottom. The end point may also be determined by allowing a drop of the supernatant fluid to fall on a filter paper soaked in acetic acid and potassium ferrocyanide solution. If the Fehling's solution has not been completely reduced, a brown colour will be produced and more urine should be run in.

2. *Benedict's method.*—Because of the disadvantage of accurate determination of the end point with Fehling's solution this method is preferred by many.

Benedict's quantitative solution—

(a)	Sodium citrate	200 gm.	Dissolved by heat and then filtered and cooled to room temperature.
	„ Carbonate (anhydrous)	75 gm.	
	Potassium thiocyanate	125 gm.	
	Distilled water	about 600 cc.	
(b)	Copper sulphate	18 gm.	
	Distilled water	100 cc.	

Solution (b) is slowly poured into the solution (a) with constant stirring. Then 5 cc of 5 per cent potassium ferrocyanide is added to the mixture and the volume made upto 1000 cc with distilled water.

In this method the reduced cuprous oxide forms a white precipitate with thiocyanate and therefore the discharge of blue colour as the end point can be easily detected.

Method.—25 cc of Benedict's solution (quantitative) is taken in a glass flask or beaker containing about 3 or 4 gm. (approximately) of anhydrous sodium carbonate and a few glass beads to prevent bumping. The solution is heated on a flame and while boiling, diluted urine is run in from a burette (as in Fehling's method) gradually. A white precipitate forms. Urine is added until all the blue colour has disappeared. The reading in the burette is taken to note the amount of urine added.

The calculation is made thus,—25 cc of Benedict's solution is reduced by 50 mgm (0.05 gm) of glucose.

$$\frac{0.05 \times 100 \times 10 \text{ (Number of times the urine was diluted)}}{\text{Burette reading in cc}} = \text{gms of glucose in 100 cc of urine.}$$

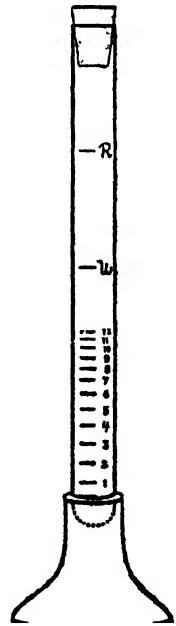


Fig. 40. Esbach's albuminometer.

Albumin (Total proteins)—*Esbach's method*—A graduated tube

called Esbach's albuminometer (fig. 40), is used for estimating albumin in urine.

Esbach's Reagent—Picric acid .. .. 1 gm.
Citric acid .. .. 2 gm.
Distilled water .. .. 100 cc.

Method.—The tube (albuminometer) is filled with urine (diluted if necessary—see below) upto the mark U and then with Esbach's reagent upto the mark R. The tube is now closed with a rubber stopper and the contents are well mixed by repeatedly inverting the tube. It is now allowed to stand undisturbed for 24 hours. The precipitated proteins settle at the bottom. The height of the deposit is read by the graduations in the lower part of the tube. The numbers indicate grams of protein per litre of urine.

Note.—(a) If the urine is alkaline it should be made slightly acid with acetic acid.

(b) If the specific gravity of the urine is more than 1010, it should be diluted sufficiently to lower the specific gravity to about 1010, before filling the albuminometer. This is necessary because the precipitated proteins will not properly sink to the bottom if the specific gravity of the urine is high.

(c) The method is not very accurate but is useful for ordinary clinical purpose. For more accurate determination other elaborate methods are necessary. Amounts less than 0·05 per cent can not be estimated by this method.

Urea.—Urea is estimated by noting the volume of nitrogen liberated from urea when a known volume of urine is brought in contact with a solution of alkaline sodium hypobromite.

For routine clinical work, a direct reading apparatus (Hind's modification of Doremus's ureometer) is used. (Fig. 41).

Reagent—This must be freshly prepared before the test.

Bromine .. .. 1 part
Sodium hydroxide solution 40% 10 parts

Method.—After closing the stop-cock the central graduated tube is filled with the hypobromite solution by pouring it in the receptacle on the left and tilting the apparatus. The side tube on the right is filled with urine upto the mark O. ½ to 1 cc or more of urine is now gradually run into the central tube by slowly turning the stop-cock. Nitrogen will be liberated, as shown by effervescence and collect at the top of the central tube. The marks on this tube represent urea in grams equivalent to the nitrogen liberated. Each small division represents 0.001 gm of urea. The amount of urine used will be shown by the graduations on the side tube. From this the percentage can be calculated.

Note.—If a large amount of urine is allowed to run into hypobromite solution suddenly, then some of the nitrogen evolved may escape through the receptacle due to vigorous effervescence. So urine should be added very slowly.

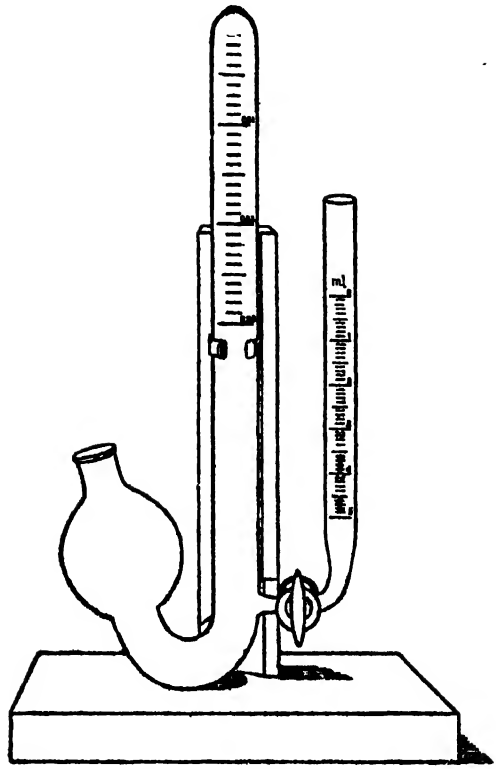


Fig. 41. Uroometer.

Examination of urinary deposits.—Urine is allowed to stand in a conical glass, or a small amount is centrifuged in a small conical tube. The deposits collect at the bottom. The supernatant fluid is drained out and a drop of the deposit is placed on a slide with a capillary pipette. This is covered with a cover-slip and examined under the microscope both with low and high power objectives.

Urinary deposits can be classified as (1) unorganised deposits and (2) organised deposits.

(1) *Unorganised deposits.*—They vary according to the reaction of the urine, and are seldom present in urine immediately when the urine is passed unless present in excessive amounts. These deposits are normally thrown out as a result of precipitation due to alteration of reaction or due to lowering of temperature of the urine after it has been voided. The various unorganised deposits have been described in table XII and shown in Fig. 42.

(2) *Organised deposits.*—These are commonly—(i) Pus cells (Leucocytes); (ii) Red blood corpuscles; (iii) Epithelial cells; (iv) Casts and cylindroids.

The organised deposits tend to disintegrate or decompose in stale urine. They should therefore be looked for in fresh urine.

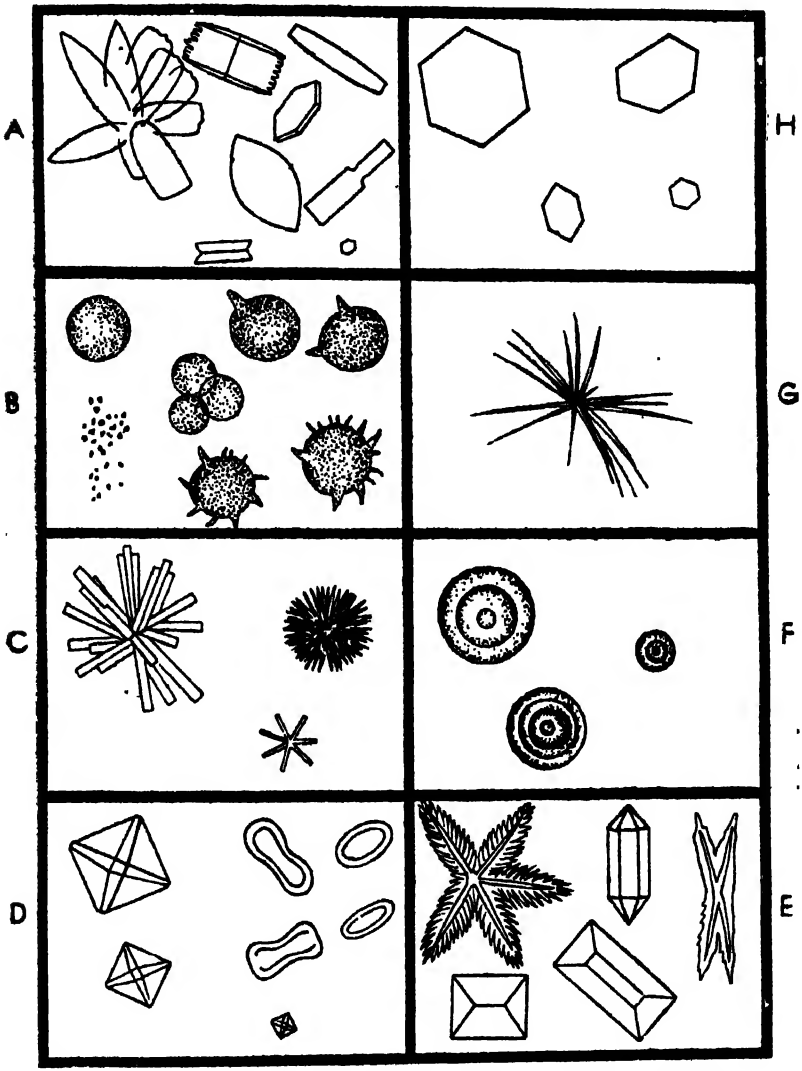


Fig. 42. Unorganised deposits in urine—A. Uric acid, B. Urates, C. Stellar phosphates, D. Calcium oxalate, E. Triple phosphates, F. Leucine, G. Tyrosine, H. Cystine.

The appearances of various casts have been shown in figure 33.

According to Addis pus cells, red blood cells and epithelial cells may be passed in urine normally upto a maximum of about 2,000,000

in 24 hours. (Roughly about 2 per field in centrifuged deposit under high power).

For significance of the presence of various formed elements in urine see page 115.

Other deposits—(i) *Prostatic threads*—These are visible to the naked eye as fine threads floating in the urine collected in a conical glass. They should be examined for pus cells and micro-organisms after smearing on slides and staining with Gram's method. They are found in chronic gonorrhœa. (ii) *Spermatozoa*—These are sometimes found in male urine. (iii) *Fat droplets*—In true chyluria fat is present in fine emulsion and the minute droplets are not visible to the ordinary high power of the microscope. When larger droplets are present, the condition is called lipuria. (iv) *Parasites and ova*—Spiked ova of Schistosoma appear in urine in vesical schistosomiasis.

TABLE XII

Unorganised Urinary Deposits.

Substance	In acid urine.	In alkaline urine.	Remarks.
1. Uric acid and urates.	As uric acid crystals and as crystalline sodium urate, or amorphous sodium, potassium and ammonium urate.	As crystalline ammonium urate.	Precipitated due to cooling of urine. Redissolved by warming or by alkalies. Deposits coloured pink or brick red due to absorption of urinary pigments, (cayene pepper deposit). Their presence is of little clinical significance. If present in suspension in urine when it is passed (lithuria), may cause irritation.
2. Phosphates.	As calcium hydrogen phosphate (stellar phosphate) in faintly acid or neutral urine.	As crystalline triple phosphate (ammonium magnesium phosphate), amorphous calcium or magnesium phosphate and as stellar phosphate.	Precipitated due to alkaline reaction of urine. Deposit may be present in urine when it is passed due to alkaline reaction or it may appear on standing due to ammoniacal decomposition of urine by bacterial action. Deposits dissolved by acetic acid or mineral acids.

TABLE XII.—*continued.*

Substance.	In acid urine.	In alkaline urine.	Remarks.
3. Oxalate.	As crystalline calcium oxalate.	As crystaline calcium oxalate.	Precipitated due to cooling of urine or may be present in suspension when the urine is passed (oxaluria) causing irritation of urinary passages and rarely haematuria. Deposit is soluble in mineral acids but insoluble in acetic acid.
4. Leucine.	As leucine crystals.	..	This is an amino acid which appears in urine in severe liver damage as in acute hepatic necrosis.
5. Tyrosine.	As tyrosine crystals.	..	Same as above.
6. Cystine.	As cystine crystals.		This is a sulphur containing amino acid which appears in urine due to an inborn error of metabolism.

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